EPIDEMIOLOGICAL ASPECTS OF ISCHAEMIC HEART DISEASE IN AMMAN, JORDAN

by

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ABSTRACT

Research into the prevalence of ischaemic heart disease and associated risk factors was carried out in Amman, Jordan between 1.4.1978 and 31.3.1979.

The study population comprised 1,444 men and 963 women aged 30-60 working in the same organization.

The objectives of this study were to determine the prevalence of ischaemic heart disease; to compare the associated risk factors and their prevalence in different age and sex groups; to initiate studies of incidence in this population; and to provide a basis for preventive programmes.

The data were collected by an interviewer-administered standard questionnaire. It included the London School of Hygiene and Tropical Medicine cardio-vascular questionnaire and questions on exercise in leisure time, diet, smoking, alcohol and oral contraceptive use. It also included questions on social and demographic variables, family and past history. Physical examination included measurement of height, weight and blood pressure. A standard resting 12-lead ECG was recorded and the tracings classified by two observers according to the Minnesota code. Plasma cholesterol and fasting blood glucose were estimated.

The methods and instructions for the interviews, examinations and measurements were standardized and based on the recommendations of the World Health Organization (Rose and Blackburn, 1968). The methods were tested for reliability and validity.

The collected data were punched on IBM cards, four cards being used for each individual. The data were edited, verified and then transferred to tapes.

Univariate and multivariate analyses were used.

The results of the study showed that there was a high prevalence of ischaemic heart disease in this cohort. They also showed a significant correlation between ischaemic heart disease and hypercholesterolaemia, hypertension, cigarette smoking, physical inactivity and type A behaviour pattern.

There were fewer cases of ischaemic heart disease among those who ate protective diet than those who ate detrimental diet. This difference was statistically significant only with ischaemic-type ECG changes without symptoms. A similar trend was observed with fat intake. The difference was statistically significant only with ischaemic-type ECG changes and ischaemic-type ECG changes without symptoms. There was, however, a significant inverse correlation between ischaemic heart disease and high fibre diet from brown bread intake.

Hyperglycaemia was associated with prevalence of ischaemic heart disease in univariate analysis, but this association may not be independent of other risk factors as it was not evident in multivariate analysis. A lack of a significant and independent correlation was also noted with body mass index.

The results of the study suggest that rapid urbanization and development and adoption of affluent and sedentary lifestyles may be associated with increased risk of ischaemic heart disease. They identify a need for preventive action and further research.

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CHAPTER I

INTRODUCTION

There is considerable evidence that ischaemic heart disease* (IHD) is multifactorial in origin and that most causative factors are environmental, the risk being increased in those communities which have adopted an affluent and sedentary life style.

IHD has been described (Morris, 1957) as having become a modern epidemic for those over middle-age, and undoubtedly, the morbidity and mortality ascribed to IHD has increased rapidly in most developed countries around the middle of this century. The mortality due to this disease has declined in the U.S.A. (Walker, 1974, 1977) and has been fairly stable in the U.K. (Marmot et al., 1978) in recent years. Although this is reassuring, nevertheless the death rate from the disease is still high, its cost to society is continuing to rise, and often its first sign is also the last.

While the establishment in developed countries of intensive care units and coronary ambulances has saved

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* The term "ischaemic heart disease" is used in the ICD 8th Revision to cover the conditions previously described as "arteriosclerotic heart disease, including coronary disease" (ICD 6th/7th Revisions). "Coronary heart disease" (CHD) is used nowadays synonymously with these two previous terms. In this thesis the term "ischaemic heart disease" is used as it conforms with current ICD usage.

a few fortunate individuals, this has had little effect on the overall morbidity and mortality.

Two arguments have been raised against coronary care units: first, that they do not, in fact, lower hospital fatality rates from myocardial infarction below those that can be achieved with ordinary hospital care; and second, that even if they are effective in this regard, their overall impact is limited because the bulk of coronary heart disease mortality occurs outside the hospital. This controversy has been extensively reviewed by Bloom and Peterson (1973), Rose (1975) and Stross et al. (1976).

The work of cardiovascular epidemiologists has identified a list of risk factors that predispose to the development of IHD, many of which appear to be preventable.

Extensive research has been undertaken on the relationship between IHD and its antecedents in western populations. Nevertheless, the relationship is clouded by controversy on the role of diet, smoking and behaviour, and uncertainty on whether the risk factors important in western populations are equally applicable in non-western societies.

In western countries elevated cholesterol, hypertension and cigarette smoking have been demonstrated as the major coronary risk factors and appear to be almost equally important in determining coronary risk (The Pooling Project Research Group, 1978). However, in the Seven Country Study (Keys, 1970) it was the mean serum cholesterol

which determined whether the different populations in these countries were high or low in IHD incidence. The two other factors - cigarette smoking and hypertension - occurred to a variable extent in all populations, so were unlikely to account for the large difference in IHD incidence. Also the cholesterol levels may not be the only determinant of national differences, for instance, Scots and Swedish are said to have similar cholesterol levels but their coronary mortality rates are different (Pedoe, 1979).

IHD is thought to be more common in developed countries than in developing countries. Most of this evidence has been derived from contrasts between comparatively affluent and primitive communities. Furthermore, the prevalence and incidence rates in developed countries have been based on extensive research while in developing countries factual information on these parameters is lacking. Most of the data available from the latter countries are either based on studies conducted in the 1950s and 1960s or, on data of questionable validity such as mortality statistics.

There are large variations in the stage of development and life style between different developing countries. Most of these countries have undertaken ambitious development plans and some, the oil producing countries in particular, have a per capita income which now exceeds that of many developed countries.

Differences in income and life-style also exist between sub-groups within a country. In Jordan for example there has been a rapid growth of a middle class with a predominantly westernized life-style.

In view of these changes it can be assumed that there are differences in the frequency and distribution of IHD amongst developing countries; and an increase in these indices has probably occurred. Urban areas experience more coronary artery disease than do rural areas. Rates go up in areas that are being urbanized, even among those people who were originally there and around whom urbanization is occurring (Tyroler and Cassel, 1964).

As Jordan is a country which is becoming developed, the aspects related to the distribution and determinants of IHD and its possible prevention require study.

As no work has been done on the factors associated with this condition in Jordan, this study has considered most of the parameters that have been shown to correlate with the incidence of this disease in developed countries.

Without overlooking the limitations of frontline research undertaken in a developing country and the biases inherent in cross-sectional data, there are advantages of a study such as this in Jordan. It does provide much needed information on IHD in a country which has recently undergone rapid developmental change. It might also clarify the uncertainties about the relationship of IHD

and its antecedents in a non-western culture.

It is hoped that the present study will form the basis for a prospective study dealing further with the antecedent factors, the incidence of the disease and its mortality rates amongst this population.

CHAPTER II

REVIEW OF LITERATURE

The morbidity and mortality rates of IHD and the factors which are thought to be conducive to the development of this disease have been amongst the most widely researched problems in medicine.

The purpose of this review is to attempt to summarize the frequency of IHD in various populations, to identify established associated risk factors, to provide basis for comparison and to consider the achievements that may contribute to possible prevention of this disease.

Not all the factors considered in the literature review will be examined in detail in the data presented in this thesis. It is hoped, however, that this review will form a basis for further analysis of the data from this study and will serve as a reference for the subject for others entering this field especially in Jordan.

Some Historical Aspects

Although it may be assumed that IHD has been in evidence since the beginning of human civilisation it was not until the second half of the 18th century that IHD was lucidly but definitively described. In 1768, Heberden described the disease in a manner which has been repeatedly

confirmed but little improved upon until the 20th century.*

Hunter described his own angina pectoris, in 1794, and following his death in that same year, his brother-in-law, Home, reported the post-mortem appearance of the coronary arteries as being "in the state of bony tubes".

In fact the autopsy observations allowed the pathological description of atherosclerosis to precede its clinical description. Brunner, in 1727, described the necropsy findings (cited in Cowdry, 1933) in the aorta of his father-in-law as "The internal coat in several places was ruptured, lacerated and rotten like fruit ...". Then in 1775, the physiologist Albrecht von Haller, stressed the atheromatous element in the lesion, rather than the sclerotic (cited in Cowdry, 1933). In his autopsies von Haller noted two types of atherosclerotic plaques in the aorta. He described the first as soft and contained "mushy or gruel-like material", and the second which he thought to be the final state of atheromatous as "bone-like" plaque.

With the beginning of the 19th century attention was focused on the microscopic and biochemical nature of

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* "Those, who are afflicted with it, are seized, while they are walking, and more particularly when they walk soon after eating, with a painful and most disagreeable sensation in the breast, which seems as if it would take their life away, if it were to increase or to continue: the moment they stand still, all this uneasiness vanishes."

the disease. Virchow demonstrated that atheromatous plaque contains lipid, and Vogel, in 1847, showed that the atheromatous plaque contains cholesterol. Since then, advances in biochemical and pathological techniques have been employed in the study of atherosclerosis, and have led to our present-day knowledge of the structure of the atherosclerotic plaque and kinetics of blood lipids.

Returning to the clinical presentation of the disease, Harrick, in 1912, added another clinical feature to Heberden's pre-cordial pain, and Hunter's angina with his exposition of coronary artery obstruction (myocardial infarction).

Medical practice, however, was slow to accept this differentiation. The 3rd edition of Sir James MacKenzie's "Diseases of the Heart", published in 1914, did not distinguish terminologically between the brief, repetitive attack of angina pectoris and the prolonged, severe and sometimes fatal attack, both of which were called angina pectoris. Nevertheless, a few years after the publication of Herrick's report, the concept of dividing cardiac pain into two main groups of angina pectoris and myocardial infarction, came to be accepted. As this distinction came to be recognized, the electrocardiogram was introduced and more and more terms describing the clinical picture of this disease were brought into use by medical practice. Among these terms were "preliminary

pain in coronary thrombosis", "impending myocardial infarction", "coronary insufficiency" and "atherosclerotic heart disease". Although some of these terms are still in use in some countries, they are gradually being replaced by more simple and precise terms of "coronary heart disease" and "ischaemic heart disease".

IHD PREVALENCE RATES

In the last 30 years the acknowledged importance of IHD in developed countries has prompted several national and international surveys which have considered both the frequency and the determinants of this disease throughout the world.

Initially most of these studies were designed and pursued by independent investigators and therefore various aspects of these studies are not comparable.

Some of the well-known prevalence studies are the Tecumseh Study (Table 1) and the Evans County Study (Table 2) in the U.S.A.; the Whitehall Study (Table 3) in the U.K.; the Busselton Study (Table 4) in Australia; the Chandigarh Study (Table 5) in India; and the Lawrence Taverne Study (Table 6) in Jamaica.

Among these studies different methods were used, several diagnostic criteria were employed, and a variety of data analyses and presentation were adopted.

However, in order to overcome these constraints and to compare the frequency and distribution of the

disease between countries, international studies were undertaken. Each used standardised data collection methods, analyses and tabulations, which although slightly different were comparable. The outstanding examples of these studies are the International Co-operative Study of the Epidemiology of Cardiovascular Disease, where 16 cohorts in 7 countries (Table 7) were studied (Keys, 1970, 1980), the prevalence comparisons in Europe (Table 8) where six samples in five countries were investigated (Rose et al., 1968), and the Japanese-American Comparison Study (Table 9) where three samples of Japanese origin were investigated in three different cultures (Marmot et al., 1975).

In the Comparison Studies in Europe, the prevalence rates of IHD were high in all of the samples studied. In the "Seven Countries Study" the highest rates were reported from the U.S.A. and Finland and the lowest were found in Italy and Japan. In the "Japanese-American comparisons", the lowest prevalence rates were found in indigenous Japanese and the highest were found in the Japanese in California whose IHD rates approximated those of the surrounding non-Japanese-American population. For the Japanese in Honolulu the rates were intermediate.

Although the confusing effects of differing methods and diagnostic techniques and criteria in IHD surveys cannot be totally dismissed, the existence of international differences among these rates suggests that environment and life-style may account for varying

susceptibility to this disorder.

As may be seen from Tables 1 - 9 the prevalence rates of IHD in developed countries were very high, more common in men than in women, and these rates increased with age.

In contrast to the ample research that has been carried out on IHD in developed countries, the disease in developing countries has been under-researched. Most of the literature available refers to inadequately designed case-control studies based on IHD cases admitted to hospitals where the population at risk is difficult to determine. Even the prevalence studies of the urban Indian population (Sarvotham and Berry, 1968) and of the rural Jamaican population (Miall et al., 1972) which appear to have applied sound epidemiological methods, are not without criticism. Despite the limitations of these studies they reveal that the prevalence of IHD was high and more common than had generally been believed. Furthermore, exceptionally high rates of IHD have been reported for New Zealand Maori females (Prior, 1973, 1974), and an apparent rise in IHD prevalence in China has also been reported (Cheng, 1973, 1974). In the non-European population of Cape Town, myocardial infarction which used to be rare among Bantu men has now become common (Brock, 1972). The disease has also been reported as becoming more prevalent in Nepal (Pandey, 1970), Thailand (Tangchai, 1972), Egypt (Badran and Sorour, 1972; Massoud et al., 1978), the Sudan (Hassan and Wasfi, 1972), and Kuwait (Emara and Al-Yousuf, 1977).

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AETIOLOGY AND PATHOGENESIS

The data on the aetiology and pathogenesis of atherosclerosis and IHD have been made available through extensive research using the four main methods of clinical, epidemiological, pathological and experimental animal investigations.

Clinical studies made four basic contributions to the understanding of the pathogenesis of IHD. Firstly, that serum total cholesterol levels are higher in middle-aged persons who develop IHD than the controls who remain free from the disease (Lerman and White, 1946). Secondly, that hypercholesterolaemia occurs in certain diseases such as familial hypercholesterolaemia (Boas, 1948; Slack, 1969, 1974); nephrosis (Bloor, 1917), hyperthyroidism (Epstein and Lanell, 1922), xanthomatosis (Thanhhauser, 1940) and diabetes mellitus (Rabinowitch, 1935; Joslin et al., 1971). Premature severe atherosclerosis is a common finding in patients with these diseases. Hypercholesterolaemia associated with these diseases is endogenous and is largely unrelated to diet. Thirdly, that serum total cholesterol can be lowered by dietary manipulations such as reductions in saturated fat and cholesterol intake. Fourthly, they produced observations on other risk factors such as hypertension, diabetes mellitus and obesity (Stamler, 1967). Thus they stimulated epidemiologists to ascertain these observations.

From the 1940s and extending into the 1980s a number of epidemiological studies were undertaken to assess the incidence and prevalence of IHD and to verify the risk factors associated with this disease. These were conducted either within or between populations. Examples of international studies have already been cited. The most extensive research in one country on IHD and its attributes has been carried out in the U.S.A. where three types of studies have been identified: those that sampled the whole country, such as the Health Examination Survey (HES) of 1960-62, and the Health and Nutrition Examination Survey (HANES) of 1971-74; those that sampled a whole community, such as the Tecumseh Study (1959-60) (Epstein et al., 1965), and the Framingham Study, begun in 1948 (Gordon and Kannel, 1968), and those that sampled or pooled the data of well-defined, broad-referenced groups, such as the Lipid Research Clinics Program Prevalence Study of 1971-1975, which sampled occupational and industrial groups, households and schoolchildren, and their parents (Heiss et al., 1980), and the National Cooperative Pooling Project where data from men in eight long-term U.S. Population Studies were reported (Pooling Project Research Group, 1978). The findings from five studies were sufficiently comparable to be combined in one pool, the data for the other three studies being reported separately.

The pathological studies and observations which

were initiated in the 18th century and continued through the19th century have paved the way to the understanding of the current pathology of atherosclerosis. Pathologists also studied and compared the geographic pathology of IHD with the aim of demonstrating differences between populations varying in living standards (Rosenthal, 1934; McGill, 1968), and the prevalence of atherosclerosis among young persons (Enos et al., 1955; Strong and McGill, 1962).

Pathological studies, particularly in the U.K. were also initiated to investigate whether arterial lesion was attributable to a defect in blood clotting mechanisms and the related view that the epidemiology of acute supposedly thrombotic lesion may differ from the epidemiology of slowly developing underlying lesion (Morris and Crawford, 1961; Mitchell and Schwartz, 1963). The British data also suggested that there was an inverse relationship between myocardial scars and physical activity, emphasising the importance of the collateral circulation and the need to investigate the myocardium as well as the coronary arteries (Morris and Crawford, 1958).

With regard to experimental studies in animals, including primates, those have focused on the relationship between diet, plasma cholesterol levels and the development and regression of experimental atherosclerosis.

As most of the animal experiments have been carried out on the relationship of diet to cholesterol levels and to atherosclerosis the review of the relevant literature will be included in that of diet.

RISK FACTOR CONCEPT

The research into the aetiology of IHD showed that it is associated with a variety of traits, habits or biochemical abnormalities which may either increase or decrease susceptibility to IHD incidence. Those which may precipitate the disease are called "risk factors" and those which may prevent it are called "protective factors".

In addition to age, sex and heredity, diet, serum cholesterol, hypertension, cigarette smoking, physical inactivity, type A behaviour pattern, soft water, oral contraceptives and others have all been implicated in the causation of the disease. None of these factors has been shown to be the sole or essential factor for the development of the disease. Although the risk factor concept is one of group probability, it has been criticized because in longitudinal studies not all high risk subjects at the entry examination develop IHD while some at low risk succumb to it.

Most published work has shown that hypercholesterolaemia, hypertension and cigarette smoking constitute the major risk factors in the development of IHD. The higher the levels of these risk factors, the greater the probability for a person to develop the disease. Also, their cumulative effect is intensified when all three factors are present in combination.

However, in view of the importance of the risk and protective factors, each will subsequently be discussed separately.

DIET

Data on the relationship of diet and IHD have accumulated from six types of studies: those on data on nutrition and mortality rates published by the Food and Agricultural Organization (FAO) and the World Health Organization (WHO); secular changes in diet and serum cholesterol; epidemiological studies; controlled dietary experiments in humans; analyses of autopsy findings, and animal experiments.

Analyses of WHO and FAO Data

Analyses of data on nutrition and mortality from many countries published by WHO and FAO have shown that the consumption of total fat is strongly and positively associated with mortality from atherosclerotic heart disease (Masironi, 1970; Stamler et al., 1970; Connor and Connor, 1972; Armstrong et al., 1975). Masironi has shown that when saturated fat intake is high total fat intake is also high and *vice versa*. These studies have also shown a significant association between atherosclerotic heart disease mortality rates and other

dietary factors (total calories, total protein, sucrose, meat and eggs). Furthermore, a high inter-correlation has been observed between these foods and saturated fat. Shaper and Marr (1977) consider that "these close inter-relations diminish the usefulness of this information in assigning etiological significance to specific nutrients and foods". In contrast to the lipid hypothesis, a few investigators (Yudkin, 1957, 1963, 1964, 1966, 1972; Albrink, 1963; Yudkin and Roddy, 1964; Antar et al., 1964; Albrink and Meigs, 1964) have advanced the hypothesis that high levels of sucrose intake may play a significant role in the development of IHD.

Analyses of FAO and WHO data on nutrition and mortality rates (Yudkin, 1964) on a large number of countries demonstrated a significant correlation between intake of sugar and IHD mortality rates.

Yudkin's hypothesis has been subjected to criticism by holders of the lipid hypothesis. With regard to international comparisons, Keys (1975) argues that there are countries with a high per capita consumption of sugar and of saturated fats; these countries tend to have high IHD mortality rates. Also, there are countries with low per capita sugar and saturated fat intake, which tend to have low IHD rates. When all these countries are taken together, statistical calculation shows IHD mortality to be correlated with both fat and sugar intake. Keys concludes that partial correlation

analysis shows that when sugar is held constant, IHD is highly correlated with per capita saturated fats in the diet, but when fat is held constant there is no significant correlation between sugar intake and IHD incidence rates (Keys, 1973). With respect to this matter, Stamler (1967) points out that the major sugar producing countries, such as Cuba, Venezuela and Colombia have high sugar consumption, nearly equal to that of the United States (about 40 kgm per person per annum), but their levels of consumption of meat, dairy products and fats are notably low. Despite their high consumption of sugar, these countries have low IHD mortality. Thus, for these sugar-producing countries the correlation between high sugar intake and IHD mortality does not hold.

With regard to proteins, there is some evidence that the ingestion of animal proteins may be detrimental, while the ingestion of vegetable protein may be protective against IHD.

In comparing epidemiological data relating to diet and IHD, Yudkin (1957) and Yerushalmy and Hilleboe (1957) observed that the mortality of IHD could be correlated with intake of animal protein as readily as with intake of fat.

Rizek et al. (1974) and Gortner (1975) observed that the trend of coronary mortality in the United States paralleled the ratio of animal to vegetable protein.

Recently, there has been an increasing interest in the role

of dietary fibre* in the development of IHD. Thus, the difference in IHD incidence between developed and developing countries has been postulated to be due to the difference in dietary fibre content of their foods.

In developing countries, starchy leguminous seeds, ground nuts and vegetables which are rich sources of crude fibre are common foodstuffs. Serum cholesterol levels have been found to be low and IHD uncommon in indigenous African and Asian groups (Trowell, 1960, 1972; Shaper, 1970, 1972, 1974).

However, the incidence of IHD is now rising in urban areas especially among groups who adopt Western patterns of living and diets (Sarvotham and Berry, 1968; Seftel et al., 1970; Cheng, 1974).

In developed countries common foods contain little crude fibre and less starchy carbohydrates are consumed (Trowell, 1972). In England, bread consumption decreased considerably at the turn of the present century (Hollingsworth and Greaves, 1967). The high frequency of hypercholesterolaemia and IHD in developed countries is well known.

Secular Changes in Diet and Serum Cholesterol

The recent decline in U.S. death rates from IHD

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* The term 'fibre' covers a variety of substances containing various amounts of cellulose, hemicellulose, pectins, lignins, and pentosans. Dietary fibre has been defined as the skeletal remains of plant cells that are resistant to hydrolysis by the enzymes of man (Trowell, 1972).

which began in 1964 is well documented (National Centre for Health Statistics, 1978). This decline coincided, inter alia, with a similar decline in dietary fat and cholesterol intake and in levels of serum total cholesterol over the same period (Beaglehole, 1979).

Although it is very difficult to exclude the influence of methodological variation on the observed trends, the correlation between dietary changes, decline in serum cholesterol and reduction in IHD mortality, lends support to the diet-cholesterol-IHD hypothesis.

Epidemiological Studies

The association between diet and IHD has been studied in diverse population groups. These include population samples with different habitual diet patterns, studies in special groups such as vegetarians and primitive people, and studies in migrants.

Studies on Population Samples

Three of the several studies which have considered the relationship between diet intake and IHD will be cited in this review.

The first is the Seven Country Study (Keys, 1970) because of its international importance and its emphasis on the effect of saturated fat intake; the second, the work of Morris et al. (1963, 1977) in which the roles of bread and fibre intake in relation to

serum total cholesterol and IHD incidence respectively were reported; and the third, the sugar intake study by Yudkin and Roddy (1964) because of the controversy it provoked over the aetiology of IHD.

Saturated Fat Intake

The Seven Countries Study (Keys, 1970) is an international prospective study of 16 cohorts in the United States, Japan, Finland, the Netherlands, Yugoslavia, Italy and Greece. The participants were about 12,000 men, aged 40 to 59 at entry. This study focused on the diet and its relationship to the aetiology of IHD. Substantial differences in the prevalence and incidence of IHD were observed among the population samples and these were associated with differences in the fat content of the diets of the populations concerned.

The highest 5-year incidence rates were recorded for men from Eastern Finland (over 120/1000 population) and the United States (over 80/1000 population) and the lowest rates were about 20 or less per 1000 for men in Corfu, Crete, Dalmatia and Japan. The amount and type of habitual diet (particularly saturated fat) varied substantially among the population samples. Finland, the Netherlands and the United States revealed high saturated fat intake, while Japan and the other three European samples revealed low saturated fat intake.

Saturated fat intakes and 5-year incidence rates

of IHD for these cohorts showed significant positive correlations. Saturated fat intake and serum cholesterol level of the cohorts were significantly correlated. Also serum cholesterol levels and IHD incidence rates were significantly correlated. Surprisingly, sugar was also correlated with saturated fat intake, serum cholesterol level and IHD incidence.

However, within cohorts there was no correlation between individual serum cholesterol measurements and the estimated nutrient intakes of those individuals.

Most of the other diet items (total calories, total fat, mono-unsaturated fat, polyunsaturated fat and total protein) were not significantly correlated to serum cholesterol level or IHD incidence rates of the cohorts.

The data on dietary cholesterol was incomplete and it was not possible to estimate any independent contribution of dietary cholesterol to either the serum cholesterol or IHD incidence.

Fibre Intake

In the Bank Staff Study, Morris et al. (1963) showed that bread consumption had a statistically negative correlation with plasma cholesterol level. Bread was an important source of dietary fibre.

Later research (Morris et al., 1977) based on a twenty year study of 337 middle-aged men, showed that men with a high energy intake had a lower rate of IHD

than the rest, and independently of this, so did men with a high intake of dietary fibre from cereals; there was no evidence that the disease was associated with consumption of refined carbohydrates.

Sugar Intake

Reference should be made to the study by Yudkin and Roddy (1964) in which they compared diet intake in patients with occlusive atherosclerotic disease (both coronary and peripheral) with controls. In this retrospective study involving 70 middle-aged men, sugar intake was reported to be significantly higher in the subjects with myocardial infarction or peripheral arterial disease compared with controls. Yudkin's claim that men who develop IHD consume more sugar than men without IHD has been refuted in all of the seven surveys on larger numbers and with better methods (Keys, 1971).

Studies in Vegetarians

Studies of the Seventh-Day Adventists* in the United States provide interesting evidence of differences

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* Seventh-Day Adventists are a conservative religious denomination who abstain from tobacco and alcohol. Almost all of them abstain from pork products and other biblically defined unclean meats and they abundantly use fruits, whole grains, vegetables and nuts. The church highly recommends, but does not require, adherence to several other life-style characteristics such as a lacto-ovo-vegetarian diet containing no meat, fish or poultry, but which does contain dairy products and eggs.

in the total cholesterol levels and IHD incidence of a religious group, classed by dietary habits as non-vegetarians, lacto-vegetarians, and pure vegetarians who consume no animal products of any type (Taylor et al., 1976; Walden et al., 1964; West and Hayes, 1968; Sacks and Castelli, 1975; Phillips et al., 1978). These studies revealed that the serum cholesterol levels of vegetarians were significantly lower than those of their non-vegetarian counterparts (Sacks et al., 1975). They also revealed that the age-standardized IHD mortality for the Seventh-Day Adventists varied systematically (Table 10). Also, the incidence of IHD has been reported to be about 40% less amongst these men than among men in the general population of the U.S.A. Furthermore, Hardinge et al. (1962) in their studies of vegetarians, reported significant inverse relationships between serum cholesterol levels and total unsaturated fats. They also reported significant positive correlations between the percentage of animal fat intake and serum cholesterol levels.

It has been postulated that the low incidence of IHD among these men may be attributed to the consumption of less meat and fat of animal origin (Walden et al., 1964).

On the other hand, it has been suggested that the low serum total cholesterol of vegetarians could also be attributed to their fibre-rich diet (Trowell, 1972).

Studies in Primitive Communities

Several studies have been conducted among people who lead primitive ways of life in bushes, highlands and other isolated rural areas (Mendez et al., 1962; Miller, 1968; Truswell, 1968; Sinnet and Whyte, 1973; Connor et al., 1978). These people subsist on a very low intake of saturated fatty acids and cholesterol in comparison to affluent Western societies and correspondingly have significantly lower serum total cholesterol levels.

In contrast, there are some exotic peoples who have low serum cholesterol values although they subsist almost entirely on foods rich in saturated fatty acids. Examples of these are the Masai and Samburu peoples (Shaper and Spencer, 1961; Shaper, 1962; Mann et al., 1964). With regard to diet-cholesterol relationship in these tribes, Keys (1975) argued that this did not disprove the diet-lipid theory, it simply meant that they differed in their cholesterol metabolism. Shaper (1972) posed an interesting question as to who should be considered as normal, the Masai and Samburu or the rest of the world.

Studies in Migrants

Several studies indicated that shifts to a 'richer' diet which follows emigration from a less affluent to a more affluent country were associated with increase

in levels of serum cholesterol and IHD incidence, prevalence and mortality rates.

In the 1950s Larsen (1957) and Keys et al. (1958) contrasted indigenous Japanese men, Japanese men living in Hawaii and Los Angeles, and Caucasians in Hawaii and Minnesota. The amount of dietary fat intake and IHD rates were highest for Caucasians in Hawaii, intermediate for Japanese in Hawaii and lowest for the indigenous Japanese.

The most notable of these studies has been the epidemiological comparisons of IHD and stroke in Japanese men living in Hiroshima and Nagasaki, Japan; in Honolulu, Hawaii; and in San Francisco Bay Area (The Ni-Hon-San Study). This study was initiated in 1965 and its findings have recently been published. Mean intakes of total fat, saturated fat, cholesterol, unsaturated fat, total protein, animal protein and simple carbohydrate were higher in the Japanese-American men than in indigenous Japanese (Kato et al., 1973; Kagan et al., 1974). The Japanese-Americans had a higher serum cholesterol level and IHD incidence, prevalence and mortality rates than the indigenous Japanese (Marmot et al., 1975; Worth et al., 1975; Robertson et al., 1977).

Conclusions from these studies suggest that the aetiology of IHD should be sought in the environmental and cultural rather than the genetic differences between people.

Trials of Diet in the Prevention of IHD

It has been shown that the frequency of IHD in a population correlates with the prevailing level of serum total cholesterol, and that the latter is influenced by the amount of saturated fat in the diet. In view of this evidence it would seem reasonable to assume that, by reducing the saturated fat intake, the IHD incidence would be lowered. Thus several trials (Table 11) have been conducted, but unfortunately the findings of these trials have been subject to criticism. The design of these trials shows that none of them satisfied all the necessary criteria in respect of duration, numbers involved and extent of cholesterol lowering achieved. Some also included subjects with IHD and thus were a mixture of primary and secondary preventive trials. The only trial (Miettenin et al., 1972) in which an unequivocal reduction of IHD mortality was achieved, also has the drawback of having been conducted on patients in two mental hospitals, rather than on ordinary people.

Controlled Dietary Experiments in Humans

Fatty Acids

The important studies of Ahrens et al. (1954), Bronte-Stewart et al. (1956), Keys et al. (1957) and Hegsted et al. (1965) demonstrated that increases in saturated fatty acids (myristic and palmitic acids) in

the diet raised serum total cholesterol levels, whereas polyunsaturated fatty acids (linoleic acid) lowered them. The effect is on β or LDL; and α or HDL are not affected (Bronte-Stewart et al., 1956; Durrington et al., 1977); and it persists if the diet is continued (Leren, 1966).

Controlled studies in adolescents (Ford et al., 1972; Vergroesen and de Boer, 1971) and children (Glueck and Stein, 1978) have also demonstrated a close relationship between plasma cholesterol levels and dietary intake of cholesterol, saturated and polyunsaturated fat (Stein et al., 1975). Both colesterol restriction and alteration of polyunsaturated/saturated (P/S) fat ratios in adolescent groups may lead to significant reduction in plasma cholesterol levels (Vergroesen and de Boer, 1971; Ford et al., 1972; Stein et al., 1975).

On the other hand, Grasso et al. (1962), Erickson et al. (1964) and Mattson et al. (1975) found the level of plasma cholesterol in metabolic studies in man to be the same regardless of the isometric structure of the dietary fatty acids.

Dietary Cholesterol

The effect of exogenous cholesterol intake on serum total cholesterol has been contradicatory and confusing. Some studies (Connor et al., 1961; Mattson et al., 1972) have shown that dietary cholesterol

increases cholesterol concentration while others (Slater et al., 1976; Mistry et al., 1976) have shown that no significant increase in total cholesterol has occurred. It seems that the effect of exogenous cholesterol differs from that of saturated fat in being smaller and more variable. The effect of the ordinary intake of eggs has also been tested, plasma cholesterols after 3 months on one egg daily (about 250 mg of cholesterol) were not significantly different from 3 months on no eggs (Porter et al., 1977).

It has been postulated that there may be a dietary threshold of cholesterol intake above which no simple linear effect of diet on cholesterol levels is exerted (Morris et al., 1963).

Thus the dietary cholesterol increased cholesterol concentrations of those persons whose base-line diets contained very low amounts of cholesterol, whereas it had no effect on those whose base-line diets already contained considerable amounts of cholesterol.

The interaction between dietary cholesterol and cholesterol concentration appears to be complicated by genetic markers. Controlled dietary experiments on Masai, who have low cholesterol levels, have shown that they differ strikingly from Western people. When Masai men were fed large amounts of dietary cholesterol, they were able to suppress endogenous cholesterol synthesis and maintain a low cholesterol level (Biss et al., 1971; Mann, 1974).

Carbohydrates

Serum total cholesterol is little affected by dietary carbohydrates. In isocalorific experiments, Dunnigan et al. (1970) and Mann and Truswell (1972) showed that exchange of sucrose for starches at 23% of dietary energy had no effect on plasma total cholesterol in healthy subjects. This is rather more than the average sucrose intake in Western countries.

Mann et al. (1973) have also shown that at 34% of total energy on sucrose, triglycerides increased by 11% and cholesterol by 5%. This was on 160 gm of sucrose which is more than most people eat daily.

Proteins

The controlled dietary experiments suggest that vegetable and animal proteins have different atherogenic effects on man. Olson et al. (1958) reported a decrease in serum cholesterol when the daily protein intake of their subjects was changed from 100 gm, mainly animal protein to 25 gm protein from cereals and vegetables. Similarly, Sirtori et al. (1977) reported that a strikingly hypocholesterolaemic effect was demonstrated by a soya protein diet (which was free of cholesterol and low in saturated fat content) as compared to a control diet having more cholesterol and saturated fat.

Fibre

Although there is increasing interest in the

role of dietary fibre or "unavailable carbohydrate", nevertheless few studies have so far shown a correlation between fibre diet content and serum cholesterol concentration. Keys et al. (1961) and Kay and Truswell (1977) showed that pectin lowers plasma total cholesterol and so do apples (Canella et al., 1962). Cereal products such as bread have been shown to reduce hypercholesterolaemia if taken in large amounts (Groen et al., 1966). Oatmeal may lower plasma cholesterol (De Groot et al., 1963; Luyken et al., 1965).

Also, it has been suggested that leguminous seeds (Keys et al., 1960; Luyken et al., 1962; Grande et al., 1965) and leguminous products such as Bengal gram (Mathur et al., 1968) lower serum cholesterol.

On the other hand, it has been suggested that the serum total cholesterol of vegetarians could also be attributed to the fibre-rich vegetables and fruits which they eat.

Analyses of Autopsy Findings in Different Countries

While the prevalence of IHD is assessed by epidemiological studies, the prevalence of atherosclerosis and the degree of its severity are assessed by autopsy studies. Comparisons of autopsy findings in different countries have shown that prevalence and severity of atherosclerosis are associated with saturated fat intake.

Early findings were reported by Rosenthal in 1934, but better evidence was produced subsequently by Kimura in 1956, and better still by McGill in 1968.

Kimura reported that in 10,000 autopsies in Japan, the prevalence of severe coronary atherosclerosis was very low compared to that seen in persons of the same age in the U.S.A. He attributed these differences to the variation in habitual intake of saturated fat and cholesterol.

However, the most notable study so far has been the International Autopsy Project (McGill, 1968). The prevalence of atherosclerosis as determined by autopsy in fifteen cities throughout the world was contrasted with their respective food consumption. A significant correlation was found between saturated fat intake and atherosclerosis. No significant association was found between sugar consumption or water hardness and severity of atherosclerosis.

Animal Experimental Studies

Ignatowski (1909) provided the first clear evidence that diet plays a role in the development of atherosclerosis. He fed meat, milk and eggs to rabbits and observed that some of the animals developed atherosclerotic lesions in their aortae. He concluded that the animal protein had injured the aortic wall, but later

experiments by Stuckey (1912) and Wesselkin (1913) provided evidence that fatty substances rather than protein in these foods were primarily responsible for the lesions. Then, Anitschkow and Chalatow (1913) succeeded in producing experimental atherosclerosis by feeding diets containing cholesterol in vegetable oil. Thus, subsequent investigators have focused mainly on cholesterol and fat feeding to produce atherosclerosis.

Therefore, in animals there is ample evidence that diets containing cholesterol and saturated fat are more atherogenic than similar diets in which the fat is unsaturated (Kritchevsky, 1964, 1970). In animal primates fed diets rich in cholesterol and saturated fat, hypercholesterolaemia and aortic and coronary atherosclerosis have been produced (Kritchevsky, 1969; Vesselinovitch et al., 1976). In addition the regression of experimental atherosclerosis has been extensively demonstrated in laboratory animals (Funch and Nielsen, 1960; Moore and Williams, 1964; Gresham and Howard, 1965; Kirtchevsky, 1969; Armstrong et al., 1970; Wilson and Hartroft, 1970). However, the studies of Lambert et al. (1958), Wigand (1959) and Malmros and Wigand (1959) showed that hypercholesterolaemia and atherosclerosis could be induced in rabbits by feeding semisynthetic diets devoid of cholesterol, and this has been confirmed by a number of investigators (Funch et al., 1960; Gresham and Howard, 1962; Moore and Williams, 1964; Kritchevsky and Tepper, 1965, 1968, 1975; Wilson et al., 1973).

Howard et al. (1965) varied the composition of basic semisynthetic diet by replacing the vitamin mixture with yeast and cabbage, the salt mixture by bone meal or bone ash, the corn starch by corn meal, the glucose, sucrose and potassium acetate by corn starch and the casein by purified soya protein. They reported that none of these alterations had any effect on the incidence or severity of atherosclerosis but they found that replacing casein by whole soya flour inhibited the hypercholesterolaemia and atherosclerosis observed in rabbits fed semisynthetic diets.

Carroll and Hamilton (1975) studied the effects of cholesterol-free semisynthetic diets on serum cholesterol levels in rabbits. They fed a number of proteins of animal and vegetable origin and found the latter type to be uniformly less cholesterolaemic. Of interest in these experiments are the observations that potato starch can completely vitiate the hypercholesterolaemic effect of casein and that butter or corn oil plus either casein or soya protein yields lower cholesterol levels than when either protein is fed in a low-fat diet.

On the other hand, Kritchevsky et al. (1968, 1973) investigated the effects of various carbohydrates in a semisynthetic cholesterol-free diet containing 14% hydrogenated coconut oil. They reported that starch was the most hypercholesterolaemic and atherogenic of

the carbohydrates used and glucose was the least. Intermediate results were obtained with sucrose and partially hydrolyzed starch. Carroll (1971) showed that the addition of glucose to commercial feed in the ratio of 1:3 had no effect on rabbit plasma cholesterol levels, while addition of casein in similar amounts produced a definite hypercholesterolaemia.

Grande (1974) has reviewed the effects of carbohydrates on serum lipid levels and emphasised the importance of separating the effects of simple carbohydrates from those of more complex ones. Among the latter sucrose and fructose may be more hypercholesterolaemic and triglyceridaemic than glucose. Kritchevsky (1976) concludes that complex carbohydrates contain various levels of non-nutritive fibre, and the fibre rather than the nature of the digestible sugar may account for the reported differences between simple and complex carbohydrates.

Experiments in which animals were fed diet rich with fibre resulted in reductions in cholesterol levels. Fahrenbach et al. (1966) showed that any number of mucilaginous gum in rat diet lowered serum and liver cholesterol levels. Pectin inhibited cholesterol-induced-atherosclerosis in rabbits (Ershoff, 1963), chickens (Fisher et al., 1964, 1966), and pigs (Fausch and Anderson, 1965).

Criticism

Comparisons of international data and autopsy findings are based on mortality statistics and diet

consumption figures for a whole country.

Collection of the data varies from country to country according to standards of medical care and stage of national development. These factors limit the comparability of the data.

In clinical studies IHD survivors are compared with healthy controls. The possibility of bias in the choice of controls cannot be ruled out and the disease might influence the risk factors rather than the other way round.

Animal and human atherosclerosis may not be comparable. The effects of polyunsaturated fats in the one may not be reproduced in the other. Animals have different lipoproteins from man and in some cases are known to respond to dietary items in a different way (Wilkens and De Wit, 1962; Truswell, 1979). The amounts of exogenous cholesterol used in animal experiments greatly exceeds human physiological levels of intake.

The Seven Country Study, the findings of which suggested that diet was the major determinant of coronary risk, will be taken as an example of an epidemiological study for this purpose. Although this study included countries with the highest and lowest IHD rates, it was criticised in that only seven countries were included and these might have supported the dietary hypothesis by chance. Dietary sugar, saturated fat, serum cholesterol

and incidence rates were all highly correlated. This might suggest that dietary sugar and saturated fat were of equal importance in the development of IHD.

Within different cohorts neither coronary risk nor individual cholesterol levels appeared to be correlated with individual diet. The last criticism weakens the diet hypothesis but may be explained by limitations of dietary data collection methods; the intra- and inter-individual variation of cholesterol measurement and variability of the effect of different diets on serum cholesterol. However the study involved countries with narrow as well as wide ranges of dietary intake.

The study was also criticised because other important dietary and psycho-social factors were not measured.

Although the design of the trials of diet and pharmacological drugs was criticised by the proponents of the diet hypothesis, their results were criticised by its opponents. Some of these trials did not provide hard data to show convincingly positive results. Furthermore, among IHD survivors, low fat (Hospital Research Committee, 1965) and soya-bean oil diets (Medical Research Council, 1968) and Clofibrate (Coronary Drug Project, 1975) did not appear to prevent recurrence of fatal heart attacks. There is also some evidence that cholesterol-reducing agents increase the hazards of gallstones and might contribute to an increase in incidence of bowel cancer (Committee of Principal Investigators, 1978).

Conclusions

Diets rich in saturated fat, or with a low ratio of polyunsaturated to saturated fatty acids are associated with IHD. Diets that are high in saturated fat are high in cholesterol. Dietary cholesterol contributes to plasma cholesterol but other dietary and non-dietary factors also affect the serum cholesterol level.

There is evidence that vegetable proteins may lower serum cholesterol level while animal proteins may have the opposite effect.

Although sugar forms a part of the calorific intake, there is no firm evidence linking sugar intake with IHD.

There is some evidence that low dietary fibre intake is associated with IHD. Fibre has also been shown to possess lipid-lowering properties.

It is worth noting, however, that despite the extensive literature available on the relationship between diet and IHD, this topic continues to provoke scientific debate. Recently Keys (1975) and Stamler (1978) repromulgated the role of saturated fat intake in the development of IHD, but the opposite view has been expressed by Mann (1978) and McMichael (1979).

On balance, it appears that there are more epidemiologists in favour than against such a relationship. There does not appear at present to be a viable alternative

to the diet hypothesis which explains the differences in IHD incidence between countries. The diet-IHD relationship has also been accepted by national and international organizations which have made recommendations on the reduction of saturated fat intake (Shaper and Marr, 1977).

SERUM TOTAL CHOLESTEROL

As the relationship between diet and IHD is presumably not direct but is through the effect of diet on serum cholesterol, the cholesterol-IHD relationship has already been considered. Nevertheless, there are a few issues which remain to be discussed.

The story of cholesterol began when Pouletier de la Salle in 1769 drew attention to a substance in gallstones that was soluble in alcohol and formed crystals upon evaporation (Bills, 1935).

Chevreul (1824) repeated de la Salle's experiments and named the substance "cholesterol" (from the Greek chole = bile, steros = solid). Later Denis (1830) reported that human blood contained cholesterol.

Vogel (1847) investigated the biochemical nature of atherosclerotic plaques and observed that cholesterol was present in these lesions. The biochemist Windaus (1910) analysed various diseased aortae and reported six times as much cholesterol and twenty times

as many cholesterol esters in atherosclerotic aortae as had been observed in normal aortae.

After these observations, extensive research was (and still is being) undertaken into the relationship between cholesterol and IHD. Recently, more interest has been shown in the cholesterol-lipoprotein-IHD relationship.

Serum Total Cholesterol Levels

Serum total cholesterol levels for children Table 12) and adults (Table 13) have been reported for many populations.

Despite limitations of inter-survey comparisons, mean total cholesterol levels for the newborn appear to be similar everywhere, while disparities in mean total cholesterol levels are found at all ages starting as early as the first year. Levels of total cholesterol in youth, however, tend to continue into adulthood.

Serum Cholesterol Levels and IHD Risk

There is strong and consistent correlation between serum cholesterol level and IHD risk.

Within populations, the risk of developing IHD rises with increasing concentration of serum cholesterol (Rosenman et al., 1967; Kannel et al., 1971; Carlson

and Bottiger, 1972; Westlund and Nicolaysen, 1972; Wilhelmsen et al., 1973; Gordon et al., 1974; McGee and Gordon, 1976; Kannel et al., 1979). However, some variations in findings have been reported. For example, an excess of IHD cases was noted in the lowest total cholesterol quintiles in the Pooling Project data (Table 14). This might be attributed to the small number of cases in these total cholesterol groups.

Between populations, strong positive relationship has been shown between the mean level of serum cholesterol and the incidence of IHD (Keys, 1970).

In emigrants, the serum total cholesterol means and distributions approximated to those of the country of immigration (Kagan et al., 1974; Marmot et al., 1975; Robertson et al., 1977).

Cholesterol-bearing Lipoproteins

Cholesterol is carried in blood plasma in five classes of lipoproteins. These are the chylomicrons, the very low density, intermediate-density, the low-density and the high-density lipoprotein fractions. The three most important systems are the low-density lipoprotein (LDL)* fraction, the very low-density lipoprotein (VLDL) fraction and the high-density lipoprotein (HDL)**

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* Also called β-cholesterol.

** Also called α-cholesterol.

fraction. Some investigators showed that there was more to be learned from these fractions than from the serum total cholesterol. Barr et al. (1951) had shown that IHD cases generally had low high density lipoprotein (HDL) cholesterol values compared with appropriate control subjects. They concluded from these studies that the most significant lipid aberration was a low HDL cholesterol level. Also, epidemiological studies have provided the bulk of evidence that low HDL cholesterol levels are associated with IHD (Oliver and Boyd, 1955; Gofman et al., 1966; Rosenman et al., 1967; Miller and Miller, 1975; Rhoads et al., 1976; Castelli et al., 1977; Gordon et al., 1977; Miller et al., 1977; Albers et al., 1978).

Similarly, it had long been recognised that animals that carry the bulk of their cholesterol in the HDL fraction were inherently resistant to the spontaneous development of atherosclerosis, and it was difficult even to induce lesions in them. On the other hand, those animals that carry more of their cholesterol in LDL fraction have long been known to be more susceptible to experimental atherosclerosis (Barr, 1953).

Most of the circulating cholesterol in the serum is normally carried in LDL. The cholesterol in this compartment correlates best with the total cholesterol. Whatever the underlying disorder, much of what has been learned in the past about the ill-effects of a high serum total cholesterol can be attributed to the associated high levels of LDL or cholesterol carried in this lipoprotein fraction (Kannel et al., 1979).

The very-low-density lipoproteins carry most of the plasma triglyceride in the fasting state. Almost all prospective studies have found a direct correlation of these particles with risk in univariate analysis (Kannel et al., 1971; Carlson and Bottiger, 1972; Wilhelmsen et al., 1973). The inconsistency is in relation to the impact of the VLDL when adjusted for the influence of cholesterol, obesity, HDL and diabetes mellitus. When this was examined prospectively, as in the Framingham and Goteberg studies (Gordon et al., 1977; Wilhelmsen et al., 1973), no independent effect was found for either VLDL or triglyceride level that showed a risk of IHD. This suggests that the VLDL correlation with risk derives from secondary associations (Kannel et al., 1979).

Criticisms

The variation in methods used for the measurement of cholesterol value and whether the measurement is done on plasma or serum constrains inter-survey comparisons.

The partition of the total cholesterol into lipoprotein fractions and the demonstration that the risk of each fraction is different from the others may render the measurement of serum cholesterol alone insufficient to study the relationship between blood lipids and IHD.

As the relationship between serum cholesterol level and IHD is continuous over a wide range, there are no firm grounds for distinguishing optimal levels for this risk factor.

Conclusions

There is a strong and consistent positive relationship between plasma hypercholesterolaemia and IHD. The risk of IHD increases with the level of plasma cholesterol and without any evident division

between normal and abnormal levels. There is also a strong positive relationship between the mean plasma level of cholesterol in the community and IHD incidence.

It has also been shown that there is a strong inverse relationship between HDL-cholesterol level and IHD. The relationship between plasma triglyceride level and IHD is inconsistent.

HYPERTENSION

Much evidence of recent years demonstrates that hypertension is one of the major risk factors in IHD, in middle-aged men and women (Morris et al., 1966; Inter-Society Commission for Heart Disease Resources, 1970; Stamler et al., 1972, 1976; Dyer et al., 1976; Rosenman et al., 1976). Hypertension has also been shown to be associated with the incidence of sudden death (Chiang et al., 1970; Dawber and Thomas, 1971; Romo, 1973). It has furthermore been reported that mortality during the first four weeks of hospital care is higher in patients with hypertension than without (Rosenbaum and Levine, 1941; Eckerström, 1951; Wright et al., 1954).

Although both systolic and diastolic blood pressures are shown to predict IHD risk (Paul et al., 1963; Kannel et al., 1969), a larger number of studies have found that the systolic is a better predictor (Paul et al., 1963; Keys, et al., 1972; Wilhelmsen et al., 1973; Kannel, 1974; Lebrach et al., 1975). Despite this,

clinical teaching (Friedberg, 1966; Harrison, 1974) considers that the diastolic is the more important.

Prevalence

The National Health Examination Survey of 1960 to 1962, which was a random sample of the United States adult population aged 18 to 79, provided data which indicated that 15% of whites and 27% of blacks had hypertension according to WHO criteria.

The prevalence of hypertension is greater among blacks than whites, as is the tendency for it to be more severe. Hypertensive heart disease occurs in blacks from three to nearly ten times more than in whites for the same age and sex groups. Also, the mortality rate from stroke and hypertensive heart disease is four to five times higher in blacks than in whites.

Effect of Age

Cross-sectional studies in most adult populations show a tendency for the mean values for systolic and diastolic pressures to increase with age, with the exception of certain communities where this is not so (Shaper, 1974).

The 1962 National Health Examination Survey data indicated that the mean systolic pressure rises slightly during early to mid-adult life, and then more rapidly over 40. The mean diastolic pressure shows a

small but steady rise up to the age of 60, falling thereafter.

The Community Hypertension Evaluation Clinic Programme, 1973-1975 indicated that mean systolic blood pressure rises with age more than diastolic blood pressure.

White prevalence rates of systolic hypertension continue to increase even over the age of 60, prevalence rates of high diastolic pressure tend to remain stable from late middle-age on.

Studies in developing countries with negro populations have suggested that blood pressure levels rise with age in a similar way to those in developed countries and that a small or no blood pressure rise may be the exception in these people, e.g. Jamaica (Miall et al., 1962), Guyana (Ashcroft et al., 1970), Nigeria (Akinkugbe, 1969) and Uganda (Shaper and Saxton, 1969).

Heredity

The hypothesis that genetic factors play an important role in determining blood pressure levels is well covered in the relevant literature.

After reviewing eight family studies, some based on history, some on blood pressure measurement, and some on diagnostic examination, Schweitzer et al. (1967) concluded that each was consistent with a hypothesis of familial aggregation in primary hypertension.

Two recent studies have an important bearing on

the interpretation of the familial aggregation data. Zinner et al. (1976) have observed this phenomenon among young children from natural families. Biron et al. (1975) have confirmed this observation for natural but not adopted children and conclude that familial aggregation of blood pressure, whether between parent and child, or between children, is mainly the result of heredity.

Further powerful support for the important role of heredity has come from studies of twins (Mathers et al., 1961; Vander Molen et al., 1970), the most definitive one involving comparisons of monozygotic and dizygotic twins (Feinleib et al., 1975). Monozygotic twins showed clearly higher correlations for both systolic and diastolic pressures than other relatives.

Salt Intake

Epidemiological studies in Polynesia, Micronesia, Africa and South America have suggested that populations who eat small quantities of salt are relatively free of hypertension (Dahl and Love, 1954, 1957; Kohlstaedt et al., 1958; Truswell et al., 1972; Oliver et al., 1975).

Evidence presented by Meneely and Dahl (1961) indicates that hypertension is common among animals and human beings who consume large quantities of salt.

When people free of hypertension migrate from a low salt to a high salt environment and adopt western ways of life, blood pressure rises with age similar to those populations who have always lived in a high salt

environment (Freis, 1973, 1976; Weinsier, 1976).

It may be concluded that chronic intake of salt in higher amounts than required may play a primary role in the development of hypertension in man.

Obesity

There is a positive association between obesity and hypertension. This association has been shown in population studies such as those of Framingham, Tecumseh (Kennel et al., 1967; Johnson et al., 1973), and Wales (Miall, 1968), as well as in cohorts from steel mill workers (Ulrych et al., 1973), aviators (Oberman et al., 1967) and college students (Paffenbarger et al., 1968).

The association of obesity and hypertension is evident in two different ways. Overweight is more prevalent in hypertensive than in normotensive individuals (Kannel and Dawber, 1973; Loggie, 1975), and normotensive obese subjects are more likely to become hypertensive (Levy et al., 1946; Kannel et al., 1967).

Stamler et al. (1975, 1976) stated that relative weight was one of the variables which had been found to contribute independently to the future development of hypertension in both cross-sectional and prospective studies.

Mineral Content of Drinking Water and Trace Metals

The 12 towns study, reported by Shaper et al. (1975), found higher rates of sudden death in towns using soft water, and noted that hypertension was a risk factor

for sudden death from IHD. They also noted that hypertension was more frequently cited in death certificates as a contributory cause of IHD deaths in towns using soft water. Diastolic blood pressures were found to average 2 mm Hg higher ($P < 0.01$) in these towns.

A possible causal relationship between cadmium and hypertension has been postulated ever since rats, rabbits and dogs were shown to become hypertensive when fed cadmium (Schroeder et al., 1966; Thind, 1972).

A cadmium concentration as low as 1 ppm in drinking water has been shown to produce systolic hypertension in rats over a 12-month period (Perry et al., 1977).

However, data from humans are inconclusive. Some researchers have reported cadmium elevations in hypertensive individuals in their urine (Perry and Schroeder, 1955), blood (Glauser et al., 1976), and kidneys (Lener and Bibr, 1971; Schroeder, 1965), while other researchers have not been able to reproduce these findings (Wester, 1973; Beevers et al., 1976; Ostergaard, 1976).

Environmental Stress

Environmental stress may be defined in terms of socio-economic levels, crime rate, residential change and overcrowding.

Blacks living in areas with low environmental stress had less hypertension than those living in high stress areas (Harburg et al., 1970).

Also there was less hypertension among rural than urban Zulus (Gampel et al., 1967); and of the latter, more of those who held to traditional cultural practices and were unable to adapt successfully to the demands of urban living were hypertensive (Scotch, 1960).

In experiments with rats in which isolation was followed by vigorous interactivity and sustained competition for territory, hypertension occurred in murine strains not naturally hypertensive (Henry et al., 1975).

The blood pressure of normotensive individuals (Nestel, 1969) as well as of persons with early, (Baumann et al., 1973), labile or persistent hypertension (Shapiro et al., 1972; Ulrych, 1969) rises when they are emotionally or physically under stress, but the rise is considerably greater in those with hypertension.

The earlier onset of hypertension among air traffic controllers (mean age 41) as compared with their subordinates (mean age 48) has been attributed to the greater stress inherent in the air controllers' duties (Cobb and Rose, 1973).

Personality Traits

Reactive neurosis (Kidson, 1973), suppression of emotions (Pilowsky et al., 1973), repression of anger, and conflicts between passivity and aggression have all been remarked in hypertensives (Ostfeld, 1967).

High systolic values also correlate with the

length of episodes of anxiety and agitation (Heine and Sainsbury, 1970).

Later Development of Hypertension

Blood pressure level is by far the strongest factor predisposing to future hypertension. Even in the very narrow range of borderline blood pressure readings, higher initial blood pressure levels lead to more future hypertension (Miall, 1971; Stamler et al., 1975).

In several studies (Sokolow et al., 1966; Stumpe et al., 1976) patients with borderline hypertension became more hypertensive than their normotensive counterparts.

Tachycardia in itself is an unreliable indicator of future hypertension (Paffenbarger et al., 1968; Stamler et al., 1975). Far more important than its own importance in the prediction of hypertension is the diagnosis of whether the tachycardia is a benign condition or not. However, tachycardia carries higher risk for future hypertension even if the subject has normal blood pressure reading (Levy et al., 1945).

The observation that the prevalence of hypertension increases with age goes with the view that hypertension may develop in susceptible individuals the more they are exposed to an adverse environment (Stamler, 1976).

Also, the absence of a similar age-linked rise in the blood pressure of certain populations (Truswell et al., 1972; Oliver et al., 1975; Shaper, 1974) and the

increased frequency of hypertension and its complications in Westernized migrants from developing countries (Scotch and Geiger, 1963; Braxton, 1970; Cassell, 1974) bears out this view.

Criticisms

The measurement of blood pressure is influenced by many variables such as lability, arm circumference and cuff width, type of instrument used, examiner-subject interaction, observer error and whether the examinee is under anti-hypertensive treatment or not.

There is controversy over whether systolic or diastolic pressure is more important as a risk factor for IHD. Physicians consider that the latter is more important while epidemiologists favour the former.

Another controversy is whether change in blood pressure after its initial measurement may influence its ability to predict subsequent IHD incidence or not.

The relationship between arterial hypertension and IHD appears to be a quantitative one and therefore there is little evidence of a numerical division between harmless normotensive and hypertensive blood pressure

Antihypertensive treatment has had surprisingly little effect on either the incidence (Lew, 1973) or prognosis of IHD (Smirk and Hodge, 1963).

Conclusions

There is a strong and consistent relationship between arterial hypertension and IHD. The severity of coronary atherosclerosis is associated with the duration and degree of raised blood pressure.

Both raised systolic and diastolic blood pressures have been shown to be associated with IHD, but the association is stronger with systolic than with diastolic pressure.

SMOKING

The striking association, now generally conceded to be causal between cigarette smoking and cancer of the lung for some time overshadowed the dramatic excess of deaths from all causes in cigarette smokers. It was not until 1954 that the large prospective study of middle-age white American men by Hammond and Horn showed that this excess of deaths is due largely to IHD. Four years later their second report confirmed these initial observations (Hammond and Horn, 1958). A few years later these observations were strongly supported by the Albany and Framingham Studies (Doyle et al., 1962).

Then, one of the largest prospective studies (Hammond, 1966; Hammond and Garfinkel, 1969) including about one million men and women aged 40 to 84 at entry

produced data which showed that for sex and age group, IHD mortality rate increased with intensity of cigarette smoking, the younger the age-group, the higher the relative risk.

Subsequently, the data of the final report of the National Cooperative Pooling Project (1978) which presented the data of several longitudinal studies on the incidence of IHD in middle-aged men, showed that for men smoking cigarettes at initial examination the risk of developing or dying from IHD was consistently higher than those who have never smoked, or who gave up smoking. Risk also increased with the number of cigarettes smoked daily.

On the other hand, in international data comparisons, a significant correlation was found between average per capita consumption of cigarettes and IHD mortality rates for middle-aged men and women of developed countries. It is interesting to note that in one of these studies the correlation coefficients between cigarette smoking and IHD mortality rate were higher for women than for men (Stamler et al., 1970).

Consistent with the findings of the epidemiological and international data comparison studies were those of the autopsy studies. Their results confirmed the increased prevalence of severe atherosclerosis in those who were heavy smokers, prior to their death, compared to those who had never smoked (Auerbach et al., 1965, 1977; Sckétt et al., 1968; Strong et al., 1969; Strong and Richards, 1976).

However, heavy cigarette smoking in populations in which serum cholesterol concentrations are low and atherosclerosis rare, appears to have little effect on the risk of IHD (Keys, 1970; Gordon et al., 1974; Strong and Richards, 1976).

With regard to giving up smoking, all reports show a reduction in the risk of mortality from IHD in those who stopped smoking. Doll and Hill (1964) and Hammond (1966) have found that a fall in mortality is evident within one year of stopping and that it takes ten years for the death rate of ex-smokers to approach that of non-smokers. More recent reports also showed that the lowering of risk of IHD by cessation of smoking was most encouraging (Ball and Turner, 1974; Gordon et al., 1974; Doll and Peto, 1974).

Mechanisms

The main constituents of tobacco smoke thought to affect the heart are carbon monoxide and nicotine.

The present consensus is that carbon monoxide in inhaled smoke, is most likely the injurious agent. Carbon monoxide has a greater affinity for haemoglobin than does oxygen, and at the low oxygen tension prevailing in the capillaries, hinders the release of oxygen from haemoglobin (Landow, 1973). Carboxyhaemoglobin concentrations of 5 - 15% commonly are encountered in cigarette smokers and can exert a powerful hypoxiating

effect (Inter-Society Commission for Heart Disease Resources, 1972; Wald et al., 1973; Lefkewitz, 1976). Experimentally, the continuous inhalation of low concentrations of carbon monoxide damages arterial endothelium and increases permeability to lipids (Astrup et al., 1967; Thomsen, 1974; Davies et al., 1976; Stender et al., 1977). On the other hand, nicotine inhaled simultaneously with carbon monoxide stimulates catecholamines secretion and thereby increases the work of the heart and may further jeopardize the ischaemic heart (Cryer et al., 1976; Lefkewitz, 1976). Furthermore, the mobilized catecholamines may recruit platelets and significantly enhace their stickiness, thus increasing the potential for endothelial damage and for obstructing thrombus formation on ulcerated atheromatous plaques (Levine, 1973).

Relation to Other Risk Factors

In the Pooling Project (1978), when the three risk factors, blood pressure, serum cholesterol and cigarette use were considered simultaneously by applying a multiple logistic model, it was further demonstrated that consistently the relationship of each of these factors to coronary proneness was an independent one. Also, in combination, they related to risk over a wide range than did any one factor considered singly. Thus observed relative risk was about 4 to 1 when the highest quintile of risk was compared to the lower tow; and about 6 to 1 when the highest quintile was compared to the lowest.

With regard to the relation of cigarette smoking to social class, it appears that smoking habits are now changing. Smoking is now more common in people from the lower social classes than in those of higher social classes (Ashford et al., 1961; Fletcher et al., 1970; Khosla and Lowe, 1972; Holme et al., 1976; Morris, 1979). Also, smoking among British physicians has decreased significantly (Doll and Hill, 1964; Royal College of Physicians, Smoking and Health Now, 1971) and the worsening IHD mortality of social classes IV and V in England and Wales correlated with relatively more smoking (Marmot et al., 1978).

The weights of ex-smokers have repeatedly been shown to be higher than those of smokers (Ashford et al., 1961; Kopezynski, 1972; Seltzer, 1974; Gordon et al., 1975) and it is common to put on weight after smoking is stopped (Glauser et al., 1970), but it seems that the weight gain is usually limited, and the effect on the patient's risk profile is small compared with the benefit of stopping smoking (Mann, 1974; Pedoe, 1979).

Smokers often have been reported to have higher average serum cholesterol values than non-smokers (Karvonen et al., 1959; Thomas, 1960; Pincherle, 1971; Schwartz et al., 1971; Van Houte and Kesteloot, 1972; Dales et al., 1974; Tuomilehto et al., 1978).

In general, the association between cigarette smoking and elevated blood pressure has been inconsistent. In some studies it was found to be negative (Thomas, 1960;

Higgins and Kjelsberg, 1967; Larson and Silvette, 1971; Pincherle, 1971; Seltzer, 1974; Kesteloot and Van Houte, 1974) while in others the association was not maintained when other factors like obesity were controlled (Dawber et al., 1967; Tuomilehto et al., 1978). However, in other studies a positive association between smoking and blood pressure level has been found (Dawber et al., 1959; Jenkins et al., 1968).

There have, however, been a few studies in which the association between physical exercise and smoking habits have been examined. It has been shown that cigarette smoking is lower in those who do physical exercise in their leisure time than in those who do not (Hickey et al., 1975; Wood et al., 1976, 1977, 1979; Chave et al., 1978).

Criticisms

Cigarette smoking has been shown to have a stronger relationship with lung cancer than with IHD. Smokers have double the incidence of non-smokers compared with ten times as much for lung cancer.

Also this relationship is not found in all study populations. For example, in the Seven Country Study (Keys, 1970), only the U.S. railroad workers showed the smoking-IHD relationship.

Cigarette smoking tends to predict IHD less

in older persons than in young and middle-aged adults. While this may be true of other risk factors, it weakens the hypothesis that smoking precipitates acute coronary attacks (Kannel et al., 1968).

Although vascular injury from carbon monoxide, ill-effects of increased catecholamine secretion and enhanced thrombosis have been suggested, the controversy over the mechanism by which smoking contributes to the development of IHD has not been settled. Data from studies of genetically matched twins with different smoking habits have not shown the degree of association between smoking and IHD as has been found in the general population, where there is no such genetic matching (Hrubec et al., 1976; Cederlöf et al., 1977). However, in view of the small numbers in these studies of twins some investigators consider their results inconclusive (Friedman, 1977; Ramström, 1978).

Conclusions

In spite of the above criticism, a strong positive association between cigarette smoking and IHD has been shown by many studies. The risk of death from IHD in smokers is twice that in non-smokers.

The reduction in mortality for those who discontinue smoking occurs within the first year and after 10 years parallels that of non-smokers.

The criticism surrounding the controversy over mechanism and the weak relationship in old people may apply to most other risk factors.

PHYSICAL EXERCISE

Studies of London busmen and postmen by Morris et al. (1953) produced the first evidence for the protective effect of exercise on the heart. The more active bus conductors experienced less coronary heart disease than the sedentary bus drivers, and postmen less than the sedentary post office workers. The early mortality of the physically active groups was substantially less than that of the physically inactive.

Most of the studies which followed Morris's pioneer work and which were carried out in the 1950s and early 1960s showed that inactive subjects had significantly more IHD than their active counterparts. However, the majority of these studies suffered from the limitation that physical exercise was neither precisely defined nor properly assessed. In the late 1960s and early 1970s the investigators sought to define physical exercise in search of a dose-response relationship. Subsequently, two types of physical activity were categorized; the vigorous exercise in leisure time and the high physical activity undertaken in non-sedentary occupations. The former type hinged on the level of physical exercise

which is likely to reach peaks of energy output of 7.5 Kcal per minute, whereas the latter was based on jobs classed as heavy work, according to work activity measurement (Hale, 1959).

Of the numerous investigators (Table 15) who studied the relationship between physical exercise and IHD the work of two groups, in particular, has led to a better understanding of this relationship. The first group is that of Morris et al. in London studying the effect of vigorous exercise in leisure time and the second is that of Paffenbarger et al. in California studying the effect of work activity on coronary heart disease. In view of the importance of the work of these two groups the most recent two papers published by each of them will be reviewed.

Exercise in Leisure Time

Morris et al. (1973) in their study of 17,944 sedentary male executive civil servants in Great Britain between the ages of 40 and 65 over the period 1968-1970, have shown a striking difference in the relative risk of IHD between men reporting different levels of leisure activity. Men who recorded doing vigorous exercise on Fridays and Saturdays, had about one third of the incidence of IHD experienced by comparable men who did not take exercise.

A recent paper published by this study group (Chave et al., 1978) on a 20% sample (3,591 men) showed

that men who had reported vigorous exercise during the two days suffered fewer deaths from IHD through the years 1968 to 1977. There was no significant difference in mortality from other causes. Total physical activity scores were weakly related to death from IHD. Men who reported vigorous exercise smoked less than the other men.

Physical Activity in Work Time

Paffenbarger and Hale's paper (1975) on "Work activity and CHD" concerns 6,351 longshoremen in California who were 35 - 74 years old upon entry into the study. They were followed for 22 years or to death, or to the age of 75, and were classified as being high, medium or low calorie-output jobs. The high activity workers had IHD death rates almost half those found in medium and low categories; there was little difference between medium and low category workers. It has been concluded from this study that repeated bursts of high energy output established a platform of protection against coronary mortality.

In a recent paper (Paffenbarger et al., 1977) and in a sub-sample of 3,686 longshoremen they examined the correlation between IHD and coronary risk factors. The levels of job activity, heavy smoking, high blood pressure and a history of heart disease emerged as significant risk factors in predicting sudden death or delayed death from IHD.

Physical Exercise and Coronary Risk Factors

Most of the studies and the effect of physical exercise on IHD have been on serum lipids. With regard to serum cholesterol the findings are controversial. Some studies have shown that physical exercise significantly reduces serum cholesterol level (Naughton and McCoy, 1966; Lopez, 1974), while others have found no significant decrease in serum total cholesterol after an exercise programme (Fitzgerland, 1965; Mann, 1969; Bonanno, 1974). There is much consensus on the effect of exercise on serum triglycerides where highly significant decreases in serum triglycerides after exercise have been reported (Lopez, 1974; Bonanno, 1974). Recently, the effect of exercise upon plasma lipoproteins received much attention. In cross-sectional comparisons, active groups show much lower VLDL and plasma triglyceride levels than those for sedentary controls (Wood et al., 1977). When middle-aged men with mildly elevated plasma triglycerides exercised by jogging for 40 minutes per day for only four days, marked reductions in triglyceride levels were seen (Oscai et al., 1972). Plasma LDL-cholesterol levels for active men and women tend to be lower than for sedentary controls (Wood et al., 1977). Two studies have reported reductions in LDL-cholesterol in initially sedentary individuals following training programmes (Lopez et al., 1974; Weltman et al., 1978). A number of cross-sectional studies have reported significantly higher plasma HDL-cholesterol levels in very active individuals compared to their sedentary

controls (Wood et al., 1977; Ratliff et al., 1978; Erkelens et al., 1978; Hartung et al., 1978).

In summary it seems that high levels of leisure time or occupational activity are associated with plasma lipoprotein indicative of relatively low risk of IHD (Wood et al., 1979). However Morris et al. and Paffenbarger et al. showed that the relationship between physical activity and IHD is independent of other risk factors.

The vigorous exerciser tends also to show other characteristics: leanness, abstinence from cigarette smoking and had significantly lower blood pressure (Wood et al., 1976, 1977, 1979). Also Hickey et al. (1975) studied the effect of physical activity on five coronary risk factors. These were systolic and diastolic blood pressure, serum cholesterol, body weight and number of cigarettes currently smoked. They concluded that "the mean levels of the five risk factors tended to decrease with increasing leisure-time physical activity, but a similar trend was not apparent for work activity". Cooper et al. (1976) studied about 3,000 middle-aged men who reported to a clinic for cardiorespiratory fitness evaluations. These men were placed in one of five grades of physical fitness on the basis of a treadmill test performance. Serum cholesterol and tryglyceride, blood pressure, blood glucose, blood uric acid and body weight were measured for these men. All of these risk factors were significantly higher in the men with poor treadmill

performances and significantly lower in men with excellent or good treadmill performances.

In summary, the three risk factors, serum lipids, blood pressure and cigarette smoking, are lower in those who take high levels of physical activity in their leisure time than in those who do not.

Criticisms

The work of Morris et al. and Paffenbarger et al. was not without criticism. In the leisure exercise it was difficult to determine or to quantify some forms of exercise such as "vigorously getting about". Also, mis-classification could have occurred when assessment of physical exercise was made, recording only two days. However, Morris et al. minimised the effect of mis-classification by showing that a prior study had shown that the physical activities recorded for these two days correlated highly with a daily log of physical activities throughout the week. On the other hand the work activity study could have been biased by a selection process as men usually work at jobs which suit their strength, health and desires. Furthermore some of the inactive men could have become so because they were already ill. However, Paffenbarger et al. countered this criticism by showing that the coronary risk was reduced for vigorous workers even though decendents were charged against the job category that had been

held an average of six months earlier. Furthermore, the inverse association between physical activity and coronary mortality was consistent in all age classes, and persisted whether or not account was taken of job transfers that occurred.

Conclusion

Vigorous physical exercise as defined by a critical level of energy output is associated with reduced risk of coronary mortality. To produce a protective effect against IHD the level of physical exercise should stress the cardiovascular system.

BEHAVIOUR PATTERN

As early as the eighteenth century it was recognized that emotional factors play a role in relation to IHD (Heberden, 1772). Then Osler in 1892 and 1910 reported that his patients with angina exhibited an overt and characteristic behaviour pattern. Osler's observations were neglected until many years later when psychiatrists (Dunnbar, 1943; Kemple, 1945) emphasized the frequent exhibition of a strongly aggressive, hard-driving and goal-directed personality.

These personality traits received little attention, until the 1950s when Friedman and Rosenman

began to work on this subject. In 1959, they developed a rather elaborate evaluation procedure by means of which they classified men into two categories: Type A - hard-driving, impatient, a "particular action-emotion complex in which a chronic, continuous struggle is its identifiable and indispensable component"; and Type B - not so characterized, more easy going.

A large prospective study was organized in California to test the hypothesis that type A men would be unduly prone to IHD. After a two-year follow-up it appeared that the hypothesis was being sustained (Rosenman et al., 1966).

More prolonged follow-up has strengthened the statistical confidence in the differentiation and allowed examination of the relationships between behavioural types and other factors that are associated with IHD. The San Francisco group repeatedly emphasize their view that the behaviour type provides independent discrimination (Rosenman et al., 1970; Friedman et al., 1970).

As noted earlier, many investigators have reported that increased levels of risk factors such as serum total cholesterol, blood pressure and cigarette smoking are significantly associated with IHD. Despite such findings among large populations, the best combination of these traditional risk factors fails to identify most new cases of IHD. Noting that traditional risk factors account for only about half of IHD incidence in middle-aged men, Keys et al. (1972) concluded that other

variables may contribute significantly to IHD incidence.

In the search for other risk factors of IHD, a great deal of work (88 studies reported in a review by Jenkins in 1976) has been done to find out whether or not behavioural patterns do put persons at higher risk of clinically manifest coronary artery disease. In the assessment of these behaviour patterns, four measures have been employed. Three were developed in the U.S.A., the structured interview (Rosenman et al., 1975), the Jenkins Activity Survey (Jenkins, 1976) and the Framingham type A (Haynes et al., 1978), and the other in the U.K., the Bortner Scale (Heller, 1979).

The hypothesis that the type A behaviour pattern may be an additional risk factor has been supported in prospective (Jenksin et al., 1974; Rosenman et al., 1975; Theorell et al., 1975; Haynes et al., 1980) as well as in cross-sectional studies and case-control studies (Rosenman et al., 1961; Wardwell et al., 1963; Keith et al., 1965; Bengtsson et al., 1973; Thiel et al., 1973; Van Dijl, 1974; Kenigsberg et al., 1974; Shekelle et al., 1976; Haynes et al., 1978; Heller, 1979). Three of these studies (Shekelle et al., 1976; Brand et al., 1976; Haynes et al., 1980) presented data which indicated that the type A behaviour pattern was an independent risk factor for IHD.

Recently, the type A behaviour pattern has been reported to be associated with increased coronary disease

as demonstrated at cardiac catheterization (Blumenthal et al., 1975; Zyzanski et al., 1976). Blumenthal et al. (1975) found that the increasing proportion of type A patients with increasing atherosclerotic disease severity remained significant, even when age, sex, blood pressure, serum total cholesterol level and cigarette smoking were all simultaneously covaried. They concluded that "independently of traditional risk factors, behaviour pattern type A may contribute to risk of clinical CHD events via effects on the atherosclerotic process". These studies were on atypical samples - patients undergoing angiography for suspected IHD.

Also several studies have found relationships between anxiety, psychological stress, tension and emotional upset and IHD (Ostfeld et al., 1964; Lebovits et al., 1967; Bakker et al., 1967; Bruhn et al., 1969; Tibbun et al., 1972; Bengtsson et al., 1973; Thiel et al., 1973; Friedman et al., 1974). These studies suggest that angina patients are more likely to complain about somatic symptoms and to be more worried and emotionally labile than myocardial patients or persons free from IHD (Mai, 1968; Dongier, 1974). A prospective study has shown that subjects who subsequently develop angina are more likely to be hypochondrial, and anxious than non-cases (Lebovits et al., 1967).

Relation to Other Risk Factors

Friedman and his colleagues have not only suggested that type A behaviour pattern is associated with the prevalence and incidence of IHD, but is also related to the coronary risk factors.

They have suggested that ype A behaviour may raise the levels of plasma cholesterol (Friedman et al., 1958), triglyceride (Friedman et al., 1964), norepinephrine (Friedman et al., 1960, 1975), corticotropin (Friedman et al., 1972) and the insulinogenic response to glucose (Friedman et al., 1970). They have also suggested that type A may enhance the clotting of blood (Friedman et al., 1958; Friedman and Rosenman, 1959) and the sludging of erythrocytes (Friedman et al., 1964).

They also produced data which suggested that cigarette smoking and the prevalence of hypertension are predominantly found in type A subjects (Friedman, 1979).

Criticisms

Most of the research that has been published on behaviour patterns has been undertaken in the U.S.A. Therefore it is important to determine whether the type A concept is also applicable to other populations with different cultures.

Most methods used for the assessment of behaviour patterns have also been developed in the U.S.A. It remains to be seen whether the Framingham type A

questionnaire for example can be translated into other languages and used in different cultures and still retain similar assessment validity. The same applies to relationship of type A behaviour to IHD risk and whether it is similar to that found in the U.S.A.

Recently the Belgium Multifactorial Prevention Project (Kittel et al., 1978) suggested that the concept of type A behaviour pattern has sufficient cross-cultural validity to allow derivation of reliable measurements and the determination of whether type A behaviour is a risk factor for IHD internationally.

Conclusions

Type A behaviour pattern has been shown in the U.S.A. to be a significant risk factor for IHD in both men and women. In the U.K. in a case-control study, type A behaviour pattern has been shown to be associated with IHD (Heller, 1979). This association has not yet been reported in other populations.

SOCIOLOGIC FACTORS

IHD and Social Class

The literature on the influence of sociologic indices on the development of IHD has been fraught with contradiction.

Lehman (1967) observed that investigations of industrial populations, such as the Bell System (Hinkle et al., 1968) and Dupont (Pell et al., 1963), have generally found an inverse relation between occupational status and risk of IHD while studies of regional or national populations (Logan, 1952; McDonough, 1965) have usually found a positive relation.

Brummer (1967) compared mortality statistics in different countries with national data on per capita income and demonstrated a positive correlation of IHD with the standard of living.

Antonovsky (1968) reviewed many morbidity and mortality studies from the U.S.A. and U.K. He found that most studies showed large differences between social classes, but that there was no consistent gradient. Antonovsky concluded that the association of IHD with social status might be reversing itself over time.

Shapiro (1969), studying members of the Health Insurance Plan of New York, found white-collar workers to have higher rates of IHD than blue-collar, but the differences approached zero when the series was controlled for physical activity.

Studies conducted in India (Malhotra, 1967; Sarvotham and Berry, 1968) showed a consistent gradient of rates by social class, with upper classes having the high rates and lower classes the lowest.

In Chile, Viel et al. (1968) used autopsy data to study the correlation of IHD with social class. They

reported that the percentage of the coronary arteries covered by fibrous plaques was higher among men of high socio-economic status and among men who did intellectual work as contrasted with manual workers.

Marmot et al. (1978) analysed the mortality trend over 40 years in England and Wales and showed that mortality from IHD had become progressively more common in working-class men and women than in those from the middle and upper classes.

Marmot et al. (1978) showed a clear inverse relationship between grade of employment and IHD mortality of male civil servants working in London. Men in the lowest grade (messengers) had 3 - 4 times the IHD mortality of men in the highest employment grade (administrators). Men in the lower employment grade were shorter, heavier for their height, had higher blood pressure, higher plasma glucose, smoked more and reported less leisure time physical activity than men in the higher grades. For plasma cholesterol the gradient was the other way round.

Relation to Coronary Risk Factors

The high levels of serum cholesterol that have previously been found to be associated with high socio-economic status (Keys et al., 1958; Howell, 1970) are today mainly associated with lower socio-economic status (Holme et al., 1976; Tuomilehto et al., 1978).

Also men with lower socio-economic status tend to smoke more (Ashford et al., 1961; Fletcher and Horn, 1970; Khosla and Lowe, 1972; Holme et al., 1976). In both sexes, non-smokers tend to be heavier than smokers, and the difference increased progressively from Social Class I through to Social Class V (Kohsla and Lowe, 1971, 1972; Howthorne et al., 1979). Higher blood pressure at all ages, and substantially greater prevalence of hypertension have been found in blacks than in Caucasians in the United States (Comstock, 1957; NHES, 1960-1962; Heyden et al., 1969; Boyle, 1970). It is probable that this is at least partly explained by genetic factors (Boyle, 1970). However, a gradient in blood pressure has been found in relation to socio-economic status, for both blacks and whites with lowest pressures in the highest socioeconomic status and highest pressures in poorest rural groups (Langford et al., 1968). Also blood pressure tends to be higher in rural than in urban populations in England (Reid et al., 1966) and Jamaica (Miall and Cochrane, 1961).

Upper social class people also take more exercise during leisure time than do low social class people (Holme et al., 1976; Morris, 1979).

Acculturation

The process of acculturation appears to be an inevitable outcome of contact between a traditional culture and the western civilization. The assimilation into the

dominant culture of persons from traditionally oriented societies affects most aspects of life.

Evidence of the importance of acculturation has been obtained from studies of primitive people who become town dwellers and from studies of immigrant groups who have moved from areas of low to high IHD incidence where these people have lived long enough in the new culture to reach middle age and to adopt local habits, they appear to have developed similar coronary rates and risk profiles (Page et al., 1974; Marmot et al., 1975; Robertson et al., 1977). The immigration has largely been from poor to affluent countries so that the effects of reverse process are less well observed.

Criticisms

Research findings on the correlation between social class and IHD have been contradictory and inconsistent.

There is considerable variation in the social class structure between and within countries. This has been confounded by increased social class mobility as a result of opportunities for better education and wider availability of jobs.

Although several studies have shown that social class relates to IHD incidence, few have analysed the effects of intervening variables which account for these associations.

Conclusions

There is a social gradient in the incidence and prevalence of IHD.

It appears that the disease tended in the past to be more common in the upper social classes than the lower. However, this relationship appears to be changing and there is a tendency for IHD incidence and prevalence to decrease with improved socio-economic status.

HYPERGLYCAEMIA

Several studies have demonstrated that patients with diabetes mellitus experience excess of morbidity and mortality from IHD (Pell and d'Alonzo, 1970; Kessler, 1971; Keen and Jarrett, 1975; West, 1978). While this is true in western countries, there is evidence that this is not so in some non-western countries (Shaper et al., 1962; McGill, 1968; West, 1978). Furthermore, although it was possible in animal experimental work to produce both hyperglycaemia and diabetes, this was not accompanied by an intensification of atherogenesis (Stamler et al., 1959).

The question still largely unanswered, is whether diabetes is an independent risk factor for IHD in both sexes. Earlier, it was reported from the Framingham Study

(Garcia et al., 1974) that diabetes was a significant independent predictor for IHD in both men and women, but in a more recent paper (Gordon et al., 1977) diabetes was reported to predict for IHD in women only.

However, with regard to the association between IHD prevalence and blood glucose level, the literature is divided. It was suggested in both the Bedford (Keen et al., 1965) and the Tecumseh (Epstein, 1967) studies that blood glucose appeared to be related to IHD prevalence. Conversely, in Jamaica no such relationship was found (Florey et al., 1973).

The confusion about the association between asymptomatic hyperglycaemia and IHD still remains largely unsolved. Much of this confusion stems from the use of variable criteria for its definition and different methods used in its estimation.

Recently, in an attempt to resolve this confusion, the role of hyperglycaemia as a possible risk factor for IHD has been examined by studies on fifteen populations from eleven countries (The International Collaborative Group, 1979). The results of these studies considered together do not indicate a consistent, independent association between asymptomatic hyperglycaemia and IHD. The group concluded that "asymptomatic hyperglycaemia cannot be designated an established risk factor for CHD disease and the major cardiovascular diseases".

However, in these studies, glucose level was

found to be significantly associated with age, body mass index and systolic and diastolic blood pressure.

Criticisms

Inter-survey comparisons of hyperglycaemia prevalence rates are constrained by differences in methods of measurement, diagnostic criteria, population samples, and presentation of collected data.

These variations may be observed in such studies as the Tecumseh Study (Ostrander et al., 1965), the Framingham Study (Kannal et al., 1979), the Jamaican Study (Florey et al., 1973) and the American-Japanese Study (Marmot et al., 1975).

Conclusions

There is inconsistent association between hyperglycaemia and IHD.

Diabetes is associated with an increased risk of IHD when other known risk factors are present. The frequency of these factors is very high in diabetic populations.

OTHER RISK FACTORS

OBESITY

Concern about obesity continues to flourish despite the fact that there is no convincing evidence that mild or moderate obesity, in the absence of other risk factors, is a danger to health (Keys et al., 1972, 1975; Dyer et al., 1975).

In the Seven Countries Study, neither relative weight nor obesity was an independent risk factor for JHD (Keys et al., 1972). In the Pooling Project studies (The Pooling Project Research Group, 1978), relative weight was less strongly and consistently related to risk of non-fatal myocardial infarction and coronary mortality. The relationship was statistically significant only for the 40 - 49 age group of 5-Pool cohort, not at older ages. However, data from the Seven Countries Study (Keys et al., 1972) and the Framingham Study (Gordon and Kennel, 1973) indicated that overweight may contribute to the risk of developing angina pectoris.

On the other hand, autopsy studies have not found any significant correlation between adiposity and the degree of atherosclerosis in coronary arteries (Wilkins et al., 1959; Spain et al., 1963). Also no relation between relative body weight and coronary artery changes was found in coronary angiographic studies in Sweden (Cramer et al., 1966).

Relation to Other Risk Factors

The finding that among subjects who are comparable in other respects, obesity does not contribute to IHD risk, should not be over-interpreted. Obesity is usually associated with the presence of other risk factors for IHD, including hypertension, diabetes mellitus, physical inactivity and raised plasma levels of cholesterol and uric acid (Kannel et al., 1967; Keys et al., 1972; Stamler, 1973). This seems to indicate that if obesity increases IHD, then it operates largely through these associated factors.

In the Framingham Study Ashley and Kannel (1974) showed a good correlation between change in body weight and change in all four coronary risk factors studied - systolic blood pressure, serum cholesterol, fasting blood sugar and serum uric acid. Also when hyperlipidaemic men were persuaded to lose weight, they experienced a striking fall in serum cholesterol as well as in fasting serum triglycerides (Leelarthaepin et al., 1974; Blacket et al., 1975).

There is also a link between obesity and smoking habits. Obesity is inversely related to cigarette smoking. Non-smokers were found to be heavier than smokers and those who gave up smoking tended to put on weight (Khosla and Lowe, 1971, 1972; Khosla, 1979; Hawthorne et al., 1979).

There is evidence that persons obese as children, teenagers or young adults are likely to remain overweight

throughout life. Moreover excess eating and sedentary life increases weight gain (Stamler, 1967).

Apparently, exercise alone is not enough to prevent weight gain; dietary control and abstinence from excessive drinking are also necessary. A cross-sectional study (Huston and Stenson, 1974) based on a British field regiment showed a 5% increase in the body mass index during the third decade of life, despite the fact that these men were engaged in active training. Eighty per cent of the men consumed an average 1000 calories per day from beer drinking, in addition to the 3500 calories diet supplied to them.

Criticisms

Obesity and overweight are not quite the same thing. While obesity implies an excessive amount of body fat, overweight implies excessive amounts not only of fat but also of muscle, bone and other tissues and these have varying densities.

For large-scale epidemiological studies there is no practical or reliable method of measuring body fat; even the measurement of skinfold thickness is subject to observer variation and where best to measure. This has led to the development of several indices derived from body weight and height which at the same time correlate with obesity. None of these indices is ideal but

Quetelet's index (weight/height^2)* is the one of choice for epidemiological purposes. It has been shown to be highly correlated with weight and consistently independent of height (Khosla and Lowe, 1967: Evans and Prior, 1969; Seltzer et al., 1970). Florey (1970) has shown that Quetelet's is a better index for males than for females.

Conclusions

The relationship between obesity and IHD is inconsistent. This relationship has been noted to be more evident with angina.

Obesity is commonly associated with raised plasma cholesterol levels, glucose intolerance and hypertension and, in the presence of these factors obesity may become a risk factor for IHD.

ORAL CONTRACEPTIVE USE

Among current oral contraceptive users, a 3 - 5-fold increase in the risk of myocardial infarction has been reported by two British case-control studies (Mann

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* The two other indices which have sometimes been used are weight/height and height/weight 1/3rd or Ponderal Index. The former is highly correlated with weight but unsatisfactory because it is also positively correlated with height. The latter has a low correlation with weight and no correlation with height.

and Inman, 1975; Man and Vessy, 1975). Similar results have also recently been reported from a case-control study in the United States (Rosenberg et al., 1979). Two prospective studies in the United Kingdom (Royal College of General Practitioners' Oral Contraception Study, 1977; Oxford/Family Planning Association Contraceptive Study: Vessey et al., 1977) have corroborated the retrospective studies. Also, the findings of these studies are consistent with the correlation found between data on oral contraceptive use and mortality trends among women from 21 countries (Beral, 1976).

Relation to Other Risk Factors

Oral contraceptives have been reported to affect all serum lipids, but their effect on the triglycerides and VLDL is most consistent and striking (Molitch et al., 1974; Meade et al., 1977; Wallace et al., 1977; Hennekens et al., 1979). This oral contraceptive-induced hypertriglyceridaemia appears to result from enhanced VLDL production stimulated by oestrogen (Rossner et al., 1971; Afolafi, 1975). In contrast to oestrogen, progestagens tend to increase the rate of clearance of triglyceride (Rossner et al., 1971; Glueck and Fallat, 1974). However, there is no clearly demonstrated independent contribution of triglycerides per se to atherogenesis in either sex in humans (Gordon et al., 1977). Thus, more important, oral contraceptives may rise the levels of serum

total cholesterol and VLDL (Meade et al., 1977; Wallace et al., 1977; Hennekens et al., 1979), but this has not been as consistently reported as in the case of triglycerides. A small, non-significant inverse relationship has also been reported between oral contraceptive use and HDL cholesterol (Hennekens et al., 1979). Also, there is evidence that 5% of women who use oral contraceptives will develop clinical hypertension within 5 years, an incidence 2.6 times greater than that of women who do not use oral contraceptives. There are also indications that the incidence may increase to 15% after 5 years of use (Kay, 1975; Kaplan, 1975).

Oral contraceptives have also been shown to alter carbohydrate metabolism. Oral and intravenous glucose tolerance is impaired and insulin secretion is increased (Wynn et al., 1966, 1969).

Oral contraceptive users have been noted to have increased thromboembolic diseases (Vessey and Doll, 1968; Sartwell et al., 1969; Collaborative Group for the Study of Stroke in Young Women, 1974; Stolley et al., 1975).

It has been reported that myocardial infarctions occur in oral contraceptive users without angiographic evidence of atherosclerotic lesions (Engel et al., 1977). This finding directed attention to the effects of oral contraceptives on various elements of blood clotting, fibrinolysis and platelet adhesiveness and raised the question of whether alterations in coagulation also play

a role. Increased sensitivity of platelets to epinephrine has been noted in both oral contraceptive users (Howie et al., 1970) and in familial hypertriglyceridaemia (Carvalho et al., 1974).

Criticisms

The use of oral contraceptives may precipitate hypertension and thromboembolic incidents.

There is controversy over whether their relationship with IHD is causal or is due to other associated risk factors.

In certain countries the validity of data on oral contraceptives is questionable. They are available without medical prescription and there is reluctance to disclose their use.

Conclusion

The use of oral contraceptives appears to contribute significantly to the development of IHD in those who have an increased risk of the disease.

ALCOHOL

Alcohol and IHD

The hypothesis that alcohol has a protective effect against IHD is at present generally supported in the literature.

Epidemiological data have indicated a negative association between consumption of alcohol and the rate of myocardial infarction. Klatsky et al. (1974, 1976) reported a larger proportion of abstainers than moderate and heavy drinkers among the 464 patients who subsequently had a myocardial infarction.

Data from the Boston Collaborative Drug Surveillance Program (Stason et al., 1976) suggested a lower rate of non-fatal myocardial infarction in patients who consumed six or more drinks per day.

Pathological studies have also indicated a negative association between consumption of alcohol and the extent of arteriosclerosis. The results from 737 autopsies done in Yalta, U.S.S.R., indicated that frequent and systematic drinking was associated with less atherosclerotic involvement of the left anterior descending coronary artery (Lifsic, 1976).

Furthermore, a statistically significant negative association was found between deaths from IHD in 18 developed countries and alcohol consumption (St. Leger et al., 1979).

However, there is some evidence (Paul et al., 1963; Wilhelmsen et al., 1973) which conflicts with the hypothesis that alcohol has a protective effect against IHD.

Alcohol and Serum Lipids

Alcohol consumption is known to influence lipid metabolism (Losowsky et al., 1963; Leiber, 1973; Avogaro and Cozzolato, 1975). However, attention has previously focused on its relation with triglycerides. Studies by Jones et al. (1965), Leiber et al. (1966), Chait et al. (1972), Leiber (1973) and Barboriak et al. (1976) have chiefly emphasized its impact on triglycerides.

Recently, data from five study populations participating in the Cooperative Lipoprotein Phenotyping Study (Castelli et al., 1977) reported that alcohol consumption was positively associated with HDL cholesterol level in all populations. Less strong but consistently negative correlations were found with LDL cholesterol. Plasma triglycerides showed a modest positive correlation with alcohol. There are other studies whose results have also shown an increase of plasma HDL after acute or chronic ingestion of alcohol (Carlson and Bottiger, 1972; Johansson and Medus, 1974).

Alcohol and Other Risk Factors

The effect of the risk factors associated with alcohol consumption may be more important than the alcohol *per se*. Alcohol has been shown to be associated with an increased dietary intake (Olin, 1966), excessive cigarette smoking (Dreher and Fraser, 1967) and hypertension (Klatsky et al., 1977).

Criticisms

There is some controversy over the relationship between alcohol consumption and IHD.

The studies which showed a positive correlation, were criticised for including too many older and heavy drinkers.

Conversely, the studies which showed an inverse correlation were criticised because the protective effect was mainly found among moderate drinkers and that high levels of consumption were not well represented.

The possible mechanisms for the effect of alcohol intake on IHD have not been sufficiently considered.

Conclusions

Despite disagreement on the correlation between alcohol consumption and IHD, on balance moderate alcohol consumption may reduce the risk of IHD.

The excess IHD mortality reported among alcohol drinkers by some studies may be partly accounted for by other associated risk factors such as dietary intake, cigarette smoking and hypertension.

WATER HARDNESS AND IHD

The study of the association of water hardness* and coronary heart disease began only a little more than two decades ago.

In 1957 a Japanese chemist, Jun Kobayashi, published a paper drawing attention to the close association of death rates from apoplexy in various areas of Japan and the acidity of river water.

Attention was drawn to Kobayashi's work by Shroeder (1958) who using Kobayashi's data, found that "perhaps an even better correlation is found with the abnormality of river water and the death rates from all heart diseases".

When he returned to the U.S.A. Shroeder investigated the relationship of water to death rates from a variety of causes on a state-by-state basis for the years 1949-1951 (Schroeder, 1960). He found significant negative correlations with deaths from all causes, all cardiovascular diseases, coronary heart disease and cardiovascular diseases and strokes.

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* Water hardness is most often expressed as the amount of calcium carbonate or its equivalent, expressed as mg. of $CaCO_3$ per litre of water or as parts of $CaCO_3$ per million parts of water (Panel on Geochemistry of Water, 1979). The four categories of hardness in common use are 0-60 ppm, soft; 61-120 ppm, moderately hard; 121-180 ppm, hard; 181 ppm or more, very hard; where hardness is expressed as $CaCO_3$.

National Comparisons

In U.S.A. significantly negative correlations between hardness and arterio-sclerotic heart disease deaths came from the research of Schroeder (1960, 1966), Schroeder and Kraemer (1974), Voors (1971) and Sauer et al. (1971, 1974).

In the U.K., confirmation of Schroeder's findings in the U.S.A. came from the work of Morris et al. (1961), Roberts and Lloyd (1972), Nixon and Carpenter (1974) and Crawford et al. (1977). However the only discordant findings from the U.K. were reported by Stocks (1973).

International Comparisons

Masironi (1970) compared mortality in three Latin American cities with soft water and three with hard water. Hypertensive heart disease death rates were considerably lower in the hard water cities, arterio-sclerotic heart disease death rates were about the same, and death rates from myocardial degeneration were strongly disparate between the sexes.

In autopsy studies Strong et al. (1968) reported that among the whites the correlation between water hardness and the mean percentage of the intimal surfaces involved with atherosclerotic lesions was negative. Among the other ethnic groups, there was a positive correlation.

At a WHO meeting findings from autopsies done in five European cities were reported (WHO Internal Document C.V.D., 1973). Three of the cities were in the U.S.S.R., one in Sweden and one in Czechoslovakia. Hypertensive heart disease death rates among persons of both sexes aged 20 to 89 years decreased almost linearly from approximately 60 per 100,000 in the city with the least hard water to approximately 20 in the city with the hardest water. There was, however, no clear relationship of water hardness to the age-adjusted frequency of atherosclerosis in the coronary arteries (Vanecek, 1976).

Literature Review

Since these pioneering studies, a number of authors have reviewed the water factor, but their conclusions have varied. Shaper (1974) and Hudson (1976) have thought that current knowledge is sufficient to take action on water treatment, while Punsar (1973), Heyden (1976) and Wolman (1976) have considered otherwise. Punsar felt that the association of cardiovascular deaths with soft water might be spurious. Schroeder (1969), Correa and Strong (1972) and Masironi (1973) have suggested that some trace element leaked from some water distribution systems was the most likely cause.

Two of the more recent authors (Neri and Johansen, 1978) favour magnesium as the element responsible for the apparent protective effect of hard water.

Relation to Coronary Risk Factors

If the hypothesis of a causal relationship between water hardness and cardiovascular disease were tenable, then the effect of water hardness might be mediated through some of the known cardiovascular risk factors such as blood pressure, cholesterol and smoking.

Blood pressure: Although it appears unlikely that the calcium content of drinking water could have any effect on serum calcium (Goodhart et al., 1973), Langford and Watson (1972) have postulated that a "low calcium intake might accentuate the hypertensinogenic effects of sodium. If this is correct, the higher sodium intake and lower calcium intake of the soft water areas should together raise blood pressure, and hypertension is a precursor of CHD deaths ..." The hypothesis that cadmium in some soft waters might also lead to renal damage and hypertension has also been raised (Schroeder, 1969).

In the U.K., one study found that blood pressure was higher in soft water areas, and also increased more rapidly with age (Stitt et al., 1973), while another found no important differences in blood pressure (Elwood et al., 1971).

In New Guinea natives living in nine villages systolic blood pressure showed a decreasing gradient with increasing calcium content (WHO Internal Document, 1973). Masironi (1977) concluded that those villages

with the softest water were nearest to sea-level. Presumably these are the most affected by Western civilization, a factor also found to be associated with high blood pressure (Henry and Cassell, 1969).

Cholesterol levels: Serum cholesterol levels have also been linked to calcium intake (Bierenbaum et al., 1973). However, one British study found higher cholesterol levels in the soft water area (Stitt et al., 1973) while another found no important differences (Elwood et al., 1971).

In comparison studies between two cities in the U.S.A. and two in the U.K. (the first of each pair being a hard water town, the second a soft water town), Bierenbaum et al. (1973) found mean cholesterol levels to be slightly lower in soft water towns.

Smoking habits: Bierenbaum et al. (1973) found no correlation between smoking and drinking water between two cities in the U.S.A. but found a marked difference between those in the U.K. hard and soft water areas respectively. Even within a single U.S.A. state smoking habits and other personal and socio-economic characteristics varied significantly in areas with different levels of water hardness (Comstock, 1971, 1978).

Associations of calcium and magnesium in drinking water with IHD: British data (Morris et al., 1961; Stocks,

1973; Elwood et al., 1977) have shown negative correlation with calcium and no correlation with magnesium. The U.S.A. data (Schroeder, 1966; Sauer et al., 1970; Voors, 1971; Sauer, 1974) found that neither calcium nor magnesium correlated significantly with IHD death rates when the other element was controlled by partial correlation (Voors, 1971).

Both Canadian (Neri et al., 1975) and Finnish (Punsar et al., 1975) data have shown a protective effect for magnesium.

Criticisms

The negative correlation between water hardness and cardiovascular mortality rates was reported in most but not all studies. In some of these studies, the lack of association might be attributed to the inadequate use of properly adjusted rates. In other studies this has been due to a limited range of water hardness. The study of small populations, the death rates of which were subject to sampling errors, was nother cause.

Although in most of the studies significant negative correlation was found, the association did not appear in any to be strong. With the probability that coronary risk factors vary between populations, such findings may be understandable.

There is a lack of a specific effect as a

considerable number of non-cardiovascular diseases were found to have a negative correlation with water hardness.

The identity and mechanism of the water factor are still not understood.

Conclusions

There is a negative correlation between water hardness and mortality from IHD, albeit one which appears to be inconsistent.

However, a significant inverse correlation has been found in U.K. studies to be with the calcium content of the water, while in the Canadian and Finnish studies this has been with magnesium instead.

FAMILY HISTORY AND GENETIC FACTORS

The familial tendency to coronary disease may be explained partly by the common environmental factors shared by the family members and partly by the known genetic component of individual risk factors associated with coronary artery disease. Children inherit both their parents' genes and living habits.

Familial occurrence of coronary artery disease has been well documented in both family (Shanoff et al., 1961; Slack and Evans, 1966) and twin studies (Harvald and Hauge, 1970; Liljefors, 1970; de Faire, 1974).

The first degree relatives of coronary patients are reported to have a 2.5 to 7-fold increase in the risk of coronary death (Slack and Evans, 1966). The age factor here is important. A family history of IHD prior to age 50 is an indicator of significantly increased risk (Stamler and Epstein, 1972).

The children of patients with familial hypercholesterolaemia have a chance of carrying the single mutant gene, of 1 in 2. Thus the early detection and treatment of affected children may contribute to preventing ischaemic heart disease (Lloyd and Wolff, 1969).

The risk of early death from coronary heart disease in men, heterozygous for familial hypercholesterolaemia (Slack, 1969) is about ten times that of men in the general population whose serum cholesterol concentration exceeds 300 mg/100 ml. The risk of IHD in female heterozygotes is also increased.

Several large surveys have reported that serum cholesterol values tend to be higher in persons with blood group type A than in persons with other blood groups, particularly type O (Langman et al., 1969; Mayo et al., 1969; Oliver et al., 1969).

Genetic factors also influence plasma cholesterol concentrations. Plasma cholesterol levels show greater concordance in monozygotic twins compared with dizygotic twins (Pikkarainen et al., 1966).

The inheritance of plasma cholesterol concentrations is greater from mother to child than from father

to child and all first degree relationships show a far greater similarity in plasma cholesterol than that between husband and wife (Schaefer et al., 1958; Johnson et al., 1965; Martin et al., 1973).

Criticism

The findings of genetic studies and studies of twins are based on small numbers which can easily become biased and may therefore be inconclusive.

Conclusions

Genetic factors can contribute to IHD either through the congenital anomalies of coronary arteries or the genetic component of individual risk factors.

There is evidence that close relatives of persons who experience IHD prior to age 50 are at an increased risk of the disease.

CHAPTER III

OBJECTIVES OF THE STUDY

1. There has been no previous research into IHD prevalence and the profile of risk factors of this disease in Jordan. Therefore, it was hoped that this study would provide the first comprehensive research into the following:
   (i) The prevalence of IHD in different strata of the workforce of the United Nations Relief and Work Agency (UNRWA) for Palestine Refugees in Amman.
   (ii) The profile of coronary risk factors in order to define those which were significantly associated with the presence of this condition in Jordan.
   (iii) A comparison of the risk factors in different age groups of males and females.
   (iv) A comparison of the risk factors between males and females who had suffered from IHD.
   (v) To lay the foundation for studies of incidence of IHD in this population.
2. It will provide a basis for formulating preventive programmes against this disease in Jordan.
3. The results of the study will contribute to serving related administrative and scientific objectives.

It will provide a basis for planning coronary care services and stimulate the execution of other specialised studies in related aspects of this disease.

4. The study will make it possible to compare results with other studies carried out in developing as well as developed countries.

CHAPTER IV

METHODS

Population

In 1979 Jordan had a population of 2,152,273 living on the East Bank; an estimated 850,000 were living on the West Bank. Amman, the capital, had a population of 550,000.

The people of Jordan are made up of three distinct groups: town dwellers, peasant farmers or villagers and pastoral nomads (Bedouin). Two-thirds of the population live in urban areas and one-third live in rural areas.

The birth rate for the year 1976 was about 50.0 per 1,000 population. The infant mortality rate for the year 1976 was 7.3 per cent of live births.

Medical Services in Jordan

These are provided by the Government medical services, UNRWA medical services and those given by private practice. They are provided through community health centres, clinics and hospitals. It has been estimated that private practice provides about 25% of these services.

In 1977 there was one doctor for every 1,430 people, one dentist for every 7,700, one pharmacist for every 4,200 and one staff nurse for every 2,500 with 17 hospital beds for every 10,000.

Study Population

UNRWA employs about 5,500 people in Jordan, 3,576 of whom are in the Amman area. The study population was made up of staff in the Amman area between the ages of 30 and 60. When those below the age of 30 and foreign staff were excluded, the eligible study population totalled 2,633 men and women.

The study was conducted during the period 1.4.1978 to 31.3.1979. The study population was chosen for the following reasons:

(a) It is an accessible population. Access to its morbidity and mortality records is readily available to the investigator.

(b) The population lives in a defined area and one for which the latest demographic data are available.

(c) The turnover of UNRWA staff is very low because of the comparatively very good employment conditions.

(d) The information collected from the participants is, as far as possible, reliable.

The study population included males and females aged 30-60 years for the following reasons:

(a) This age group has been shown to be vulnerable to IHD.

(b) It does not contain the young with very little IHD and the elderly with multiple pathology and different epidemiological features of IHD.

(c) It has been used in studies of a similar nature. This facilitates comparison.

(d) This has allowed the study of the differences and inter-relationships of risk variables between specific age and sex categories.

The staff members of UNRWA are divided into three categories: administrative staff who are largely office workers; teaching staff in the UNRWA schools and training institutions; and thirdly manual workers such as messengers, school attendants, doorkeeper-cleaners and kitchen workers.

The UNRWA Occupation Classification Manual, which is based on education and experience, specifies the academic and professional requirements for each post. The teachers are the most eduated and the manual workers the least. The administrative staff come from all levels of education. The teachers also enjoy the longest holidays compared with the other two groups.

The UNRWA staff members are well paid in comparison to the civil servants in Jordan. They receive

periodic medical examinations at intervals of not more than 3 years, often more frequently and they enjoy diplomatic immunity in relation to their work.

In recent years Jordan as a whole has seen major developments and socio-economic changes. The study population also has experienced very rapid improvements in income and standard of living.

Data Collection

Questionnaire

A standard questionnaire was used (Appendix 1). It included the London School of Hygiene and Tropical Medicine Cardiovascular questionnaire, questions on smoking, dietary intake, alcohol consumption and exercise in leisure time and the Framingham type A questionnaire. It also covered personal and demographic data, family history and previous medical history.

The questionnaire was translated into Arabic (Appendix 2) and was administered to every participant in the study by two qualified nurses.

Examination Centre

All participants in the study were interviewed and examined at Amman Specialists' Clinic. It was chosen because of the availability there of effective medical services, trained personnel and laboratory and ECG services.

Physical Examination

The standing height was measured once to the nearest 0.5 cm without shoes.

The weight was measured once to the nearest 0.5 kg. Subjects were in underclothing and without shoes. Body mass index (BMI) was computed by the formula weight divided by height squared.

The blood pressures were measured by mercury manometers. Cuffs were applied to the subjects' left arms at heart level in the sitting position. Systolic pressure was taken at the first phase (appearance of sound) and diastolic pressure at the fifth phase (disappearance of sound). Two measurements were taken and the average of the two was used in the analyses.

ECG Recording

A standard resting twelve leads ECG was taken for each participant. Room temperature was comfortable and smoking and heavy physical exercise were avoided for 30 minutes prior to the recording. Three complexes free from baseline fluctuations per lead were recorded. Longer strips were taken if arrhythmia was present. The subject's name and date of recording appeared on each complete tracing.

The tracings were classified by two observers according to the Minnesota code (Rose and Blackburn, 1968).

Biochemical Tests

Venous blood samples were taken from the participants. Plasma cholesterol was measured by Abell-Kendall method and fasting blood glucose by Hoffman's method.

Standardization

The methods and instructions for the interviews, examinations and measurements were based on the recommendations of the World Health Organization (Rose and Blackburn, 1968). The World Health Organization criteria for IHD diagnosis and International Disease Classification (ICD) code were followed.

Training

The two interviewers underwent a specific training programme to achieve uniformity in the information that was collected.

Meetings

Meetings for research workers and laboratory technicians were held at the outset and every 2 months thereafter to brief them on the project and its progress.

Feasibility

With the staff and resources available it was feasible to examine a minimum of 200 persons per month.

At this rate of examination, the study population was examined in the 12-month period projected for the examination phase of the study.

1. Reliability

Reliability, also termed 'reproducibility' or 'repeatability', refers to the extent to which similar information is supplied when a measurement is performed more than once.

Measurements of reliability were made to assess the methods and the performance of each observer, to provide a basis for quality control and to maintain interest in good working standards. They were also intended to identify systematic differences between observers' results which might have led to misleading conclusions.

Testing of methods was carried out at three stages:

i. Before the study: A trial of the methods was conducted on 20 volunteers, who were not members of the study population, since participation in the pre-test interview might have influenced the responses given in the study questionnaire. Subjects were chosen from civil servants in Jordan. Each of the interviewers interviewed and examined each subject, then systematic differences were looked for by comparing the mean and standard deviations

from the two interviewers' results.

ii. During the study: Test-retest method (within-observer comparison).

Blind Comparisons

A sample of 20 subjects was re-interviewed and re-examined by the same observer who did not have the results of the first interview and the related measurements available to him.

Replicate tests were made on the same blood specimens by the same laboratory technician using the same methods.

Coding was made by the same ECG coder of the same ECG tracings on two occasions.

Inter-observer Comparison

Reliability here was measured by the two observers performing independent interviews or measurements of the same variables on the same subjects and the findings were compared. A sample of 20 subjects was interviewed separately by the two interviewers.

A systematic sample of one-in-twenty was re-interviewed and re-examined by the study supervisor.

The results of the tests carried out before and during the study did not reveal any significant differences.

iii. At the end of the study: Each observer's results were analysed separately for their mean and standard

deviations or their prevalence rates. These will be discussed in the chapter on results.

2. Validity

Validation was carried out by:

(i) Checks from other sources

The data collected by the questionnaire on date of birth, occupation and education were checked against the information contained in the personal files of the participants. Information collected on past medical history was compared with the medical and hospital records of subjects. No differences were found between the questionnaire data and the medical and personal data.

(ii) Comparison of different diagnoses and measurements - Criterion Validity

(a) Biochemical tests: Before, and periodically during the survey, quality control was checked by local analysis of quality control samples. Freeze-dried sera from three manufacturers were analysed at random. The results are shown in Table 16. In 88 analyses of non-fasting serum cholesterol the mean coefficient of variation was 4.1%. In 114 analyses for fasting blood glucose the mean coefficient of variation was 3.5%. The coefficient of variation is a measure of precision. Values less than 8% for cholesterol and 5% for glucose are considered acceptable (Whitehead, 1977).

(b) ECG coding: ECGs were coded in duplicate according to Minnesota Code criteria, disagreements being referred to an adjudicator. This service was provided by the staff of King Hussein Medical Centre.

ECG coding of a sample of recordings of 50 subjects, consisting of both normal and abnormal ECG tracings, were sent to the Cardiac Reference Laboratory in Budapest and were checked blind. The codings done in Jordan were found to be within limits of acceptability.

(iii) Validity by follow-up (Predictive Validity)

This study constitutes a base-line for a prospective study. The prognostic significance of angina or minor ECG abnormalities will be assessed and tested by examining their relationship to the subsequent development of IHD morbidity and mortality.

Data Coding

The data on the completed questionnaire were punched on I.B.M. cards, four cards being used for each subject. The card punching was performed by the staff of the Jordan University Computer Centre. Checks were made on the range and consistency of the data. Punching and coding errors were corrected.

Data Processing and Analysis

The London University computer was used. Data

were transferred from punch cards to tapes and data files were created. Most of the analysis was done using the Statistical Package for the Social Sciences (SPSS).

As most of the variables examined were continuous, means and standard deviations were computed. Since mean differences do not provide a measure of disease frequency, prevalence rates were also computed. Direct and indirect standardization methods were used. The indirect adjustment was only applied when the figures were small.

To test for the significance of the various risk factors on the prevalence of IHD, logistic models were fitted using the computer programme GLIM (Generalised Linear Interactive Modelling).

This allowed the testing of significance of risk factors after adjustment had been made for age and sex. This was done by fitting additive models on a logit* scale that excluded the risk factors but contained the factors of age and sex. The deviation of this model from the observed data was then compared with another model that included age and sex and the relevant risk factor. The resulting figure was then compared with the percentage points in χ^2-tables and the significance of the factors obtained. To test for the independent

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* logit P = log $(\frac{P}{1-P})$ where P is the proportion of subjects with IHD.

contribution of coronary risk factors to IHD prevalence, the maximum likelihood procedure of Walker and Duncan (1967) was used and coefficient estimates were obtained. The risk factors included in this analysis were age, sex, body mass index, hypertension, fasting blood sugar, serum cholesterol, smoking, type A behaviour, physical activity and type of bread intake.

Definitions of Positive Classifications

The following categories of positive findings were defined:

1. Angina Pectoris (AP), based on answers to Section A of the standard chest pain questionnaire.

'Yes' to question 1

'Yes' to 'never hurries or walks uphill' to question 2 (if the answer to question 2 is 'never hurries or walks uphill' then the answer to question 3 should be 'yes'

'Stop or slow down' to question 4

'Relieved' to question 5

'10 minutes or less' to question 6

Site must include either any level of sternum or left chest and left arm.

2. Possible Myocardial Infarction (PMI), was defined as a positive answer to the question 'Have you ever had a severe pain across the front of your chest lasting for half an hour or more?'.

3. Symptom-positive, was regarded as being present in those who fulfilled the criteria set out in 1 or 2 above.

4. Ischaemic-type ECG signs. ECGs were classified as positive in subjects with any one or more of the following items of the Minnesota Code: Q/QS waves (codes 1.1-3); S-T depressions (codes 4.1-3); T-wave inversion or flattening (codes 5.1-3); or left bundle branch block (code 7.1).

5. Ischaemic-type ECG changes without symptoms, were classified as positive if ischaemic-type ECG changes occurred in subjects with no previous history of angina or possible myocardial infarction.

6. Suspect ischaemia was regarded as being present in those who were symptom-positive or ECG-positive.

7. Intermittent claudication (IC). Based on answers to section C of the standard questionnaire:
'Yes' to question 12, 'No' to question 13
'Yes' to question 14, 'Yes' or 'never hurries or walks uphill' to question 15, 'Yes' (grade 1) or 'No' (grade 2) to question 16, 'No' to question 17, 'Stop or slow down' to question 18, and usually disappears in 10 minutes.

8. Hypertension, was based on the WHO classification.
Normotensive: systolic blood pressure < 140 mm Hg and diastolic blood pressure < 90 mm Hg.
Borderline hypertensive: systolic blood pressure 140-159 mm Hg and diastolic < 95 mm Hg, or

diastolic 90-94 mm Hg and systolic < 160 mm Hg.

Hypertensive: systolic pressure 160 mm Hg or over, or diastolic 95 mm Hg and over.

CHAPTER V

RESULTS

For the purposes of this thesis, it is impracticable to examine in detail the association between prevalence of IHD and all the 175 variables included in the study. Only the main risk factors known to be associated with IHD are selected for study and discussion.

Response Rate

In the survey of a total of 3,576 subjects drawn from staff members of UNRWA in the Amman area, a total of 2,407 were successfully interviewed. As the study was principally concerned with people in the age range 30 - 60, all those under the age of 30 on 1.4.1978 (totalling 931) were excluded. In addition the 12 foreign staff members were excluded. Thus of a total of 2,633 staff members aged 30 - 60, 2,407 were involved in the study. The number of non-respondents was 226, giving an overall response rate of 91.4% (Table 17).

The reasons for non-response are shown in Table 18. Almost a fifth of these were unavoidable for logistical reasons. The medical records of the remainder were checked and none of them had a history of IHD. As a result, the prevalence rates shown in this study are slightly higher than in the general population.

Findings of the Interviewers

The participants were admitted to the interviews at random. There is no reason to believe that the participants selected their interviewer or the interviewer selected those whom he interviewed.

The numbers of those examined by each interviewer are presented in Table 19. Each interviewer examined about half of the participants in each age and sex group.

The percentages of angina and possible myocardial infarction obtained by each interviewer are shown in Table 20. The results are almost equal.

Age-specific means and standard deviations of the coronary risk factors measured by the two interviewers are presented in Table 21.

Age and Sex Structure

The age and sex composition of the population studied is shown in Table 22. The study population includes 60% males and 40% females. Due to the age composition of the UNRWA labour force, and the rapid growth of the Jordanian population, there are more people and hence more respondents in the lower age groups.

Prevalence of IHD

The prevalence rates of angina, PMI and ischaemic-type ECG changes are presented separately because they are different expressions of cardiac ischaemia and have different diagnostic criteria. Ischaemic-type ECG changes without symptoms are considered separately in order to study their correlation with other risk factors. This removes the influence on the latter of medical advice or awareness of having the disease.

The age-specific prevalence rates of angina, possible myocardial infarction as determined by the standard questionnaire, together with the rates of the ischaemic-type ECG abnormalities, are presented in Table 23. This table shows that prevalence rates for IHD are significantly higher for males than for females and that the rates rise with age for both sexes. These findings indicate an internal consistency of the results, since they parallel the findings of similar prevalence studies in other countries.

Prevalence of ECG Abnormalities

These are presented in Table 24 for men and in Table 25 for women.

Q/QS Findings (Code 1:1-3)

These were coded in a total of 41 men (2.8%) and 17

women (1.7%). The age gradient and sex ratio were both clearly evident. Large Q waves (code 1:1) were fewer than the other codeable Q/QS items.

Axis deviation

Left axis deviation (Code 2:1)
This was coded in 23 males (2.6%) and 6 females (0.6%). An age gradient and sex ratio were clear.

Right axis deviation (Code 2:2)
This was coded in two men but not met in women. This is partly because the code criterion for right axis deviation is stringent.

R Waves
Large R waves (Code 3:1) were coded in a total of eleven males and two females. The prevalence increased with age. Medium (Code 3:2) and small (Code 3:3) R waves were uncommon.

S T Depression
S T Depression items (Code 4:1-4) were coded in a total of 24 men and 8 women. Association with age and sex were evident except for codes 4:3 and 4:4 in females.

T wave inversion or flattening (Code 5:1-3)
These items were not uncommon. Minor or flattening of T wave formed more than half of the items. Prevalence increased with age and was higher among males.

AV conduction defect (6:1-5)

These items were rare particularly among women. First degree AV block (6:3) was observed in nine men and accelerated AV conduction was observed in four men and one woman. Complete AV block (6:1) and second degree AV block were not seen in either men or women.

Ventricular conduction defect

Left bundle branch block was observed in four men and four women. Association with age was present but not so strong. The other AV conduction defect items were rare among men and were not observed among women.

Rhythm and Rate

Premature beats were not uncommon in males but were rare in females. Atrial fibrillation was seen in three men but was not seen in women.

Low QRS amplitude (Code 9:1)

Was a common item in both sexes - association with age and sex were present.

Ischaemic-type ECG With and Without Symptoms

The age-specific prevalence rate of ischaemic-type ECG abnormalities in subjects with and without history of cardiac pain (angina or PMI) is shown in Table 26. Over half of the men with ECG changes were without cardiac symptoms, while in women those with

and those without were equal. The prevalence of ECG changes in both categories (with and without symptoms) increased with age and was more common in males than in females. About 33% of angina-positive were ECG-positive (Table 27).

Prevalence of Risk Factors

The age-specific means and standard deviation of measured risk factors are presented in Table 28.

For both males and females the mean height was highest in the 40 - 49 age-group, although the differences were not significant. Weight showed a similar pattern.

For males the highest body mass index was observed in the 40 - 49 age-group and was significantly higher than both the 30 - 39 and 50 - 60 groups. No significant differences were observed in females.

In both sexes for systolic blood pressure there was a highly significant increase with age. For diastolic blood pressure the same pattern was seen for females. However although the diastolic blood pressure increased with age in males a significant increase was only observed between the younger and intermediate age-groups.

In males the mean plasma cholesterol increased significantly with age. In females the increase was significant between the younger and intermediate age-groups but the increase between the intermediate and older

age-groups did not reach significance.

In males fasting blood glucose increased significantly with age. In females a significant increase between the younger and intermediate age-groups was observed but there was also a significant decrease between the intermediate and older age-groups.

DIET

The distribution of foods eaten and diets used are presented in Tables 29-37. For cooking, 46% of males and 41% of females used vegetable oil (mainly olive and corn oils) while the rest used either animals fats or both. For frying and spreading about two-thirds of the participants used vegetable oil while the rest used either both oil and animal fat or animal fat alone.

About half the participants ate white bread while the other half ate either brown or brown and white bread.

The majority of participants ate meat either daily or every other day. The frequency of meat-eating was high among females than young males.

With regard to vegetables the largest number of participants ate beans weekly.

Males ate less fruit than did females. About half of the females ate fruit whereas only one-third of the males did so.

Because of the difficulty of obtaining a quantitative assessment of types of food eaten, a scoring system had to be developed. The scoring system was based on a three point scale as shown in Table 38.

The scoring was made on the hypothesis that vegetable fats were protective, that a higher fibre diet from bread would be protective, that the use of vegetables and fruit daily would be protective and that excessive intake of meat might be detrimental.

For the purposes of the analysis three factors were examined:

- The dietary protective factor (DPF) which included the scores for all the dietary questions divided by their number.
- The fat protective factor (FPF) which included the scores for the three uses of fat divided by three.
- The bread factor. The bread consumption was divided into three groups: those who only ate white, those who ate brown, and those who ate brown and some white.

Tables 39 and 40 which present data for DPF show that those who ate vegetable rather than animal fat, brown rather than white bread, more beans and fruit and less meat, had less angina and fewer cases of IHD in both males and females. But there was little difference for cases who had myocardial infarction, possibly due to

a change of habit because some of them knew that they had the disease or had been given dietary advice.

Tables 41 and 42 present data for FPF. In both sexes using vegetable fat appeared to protect against IHD.

The use of brown bread alone or with some white was also protective for males and females (Tables 43 - 46).

Tables 47 - 49 compare the age-specific means and standard deviation of coronary risk factors in those whose dietary intake was postulated to be protective against IHD with those whose dietary intake was postulated to be detrimental. The age-adjusted prevalences of these risk factors are presented in Tables 50 - 52.

In both sexes the age-specific means of height showed slight differences. The age-adjusted percentages showed that men who ate detrimental diet were taller than those who ate protective diet, in women the difference was reversed.

The age-specific means of weight showed that men who ate a detrimental diet were heavier than those who ate a protective diet, while in women the differences between the means were slight.

The age-adjusted percentages of body mass index showed that in both sexes those who ate a detrimental diet were heavier for their height than those who ate a protective diet.

In men, the means of both systolic and diastolic blood pressure were higher in those who ate a detrimental diet, while in women the differences between the means were slight but appeared to show similar trends.

The prevalence of hypertension and borderline hypertension were higher in those who ate a detrimental diet in both sexes.

In men the age-specific means of plasma cholesterol were higher among those who ate a detrimental diet. In women a similar trend was noted in the younger group while in the older age groups there were slight differences. In both sexes subjects who ate a detrimental diet had a higher percentage of plasma cholesterol concentration $\geq$ 220 mg% than the other subjects.

In women the means of fasting blood glucose and the prevalence of hyperglycaemia were higher in those who ate detrimental food. In men the differences were inconclusive.

The percentage of current smokers was higher in those who ate a detrimental diet in both sexes. The percentages of ex-smokers in both sexes were roughly equally represented among consumers of both types of diet.

In both sexes those who ate a protective diet were more active than those who ate a detrimental diet.

Type A behaviour pattern was more prevalent among men who ate a detrimental diet, while in females the trend was reversed. In the case of bread, type A

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was more prevalent among men who ate brown, but in women among those eating white.

PLASMA CHOLESTEROL

In the total material, 35.4% of the men and 31.6% of the women had plasma cholesterol levels over 220 mg% (Table 53).

The prevalence rates of the various categories of IHD in both sexes correlated positively with the plasma cholesterol level (Tables 54 - 57). The age-specific means and standard deviations of coronary risk factors by plasma cholesterol level are presented in Table 58. The mean weights of males were higher in the 40 - 49 age-group at all plasma cholesterol levels, whereas in the females the mean weights showed fluctuations with age.

In males, the weight rose with increase in plasma cholesterol level in all age-groups, while in females the increase in weight with plasma cholesterol was only found in the two younger age groups.

In both sexes the BMI rose with an increase of plasma cholesterol level. In males the 40 - 49 age-group had a higher mean BMI than the older and younger age groups at all plasma cholesterol levels, whereas in the females an increase of mean BMI was only seen at a plasma cholesterol level of less than 200 mg%. In the

two higher plasma cholesterol groups the mean BMI showed a decrease with age.

Among those with a plasma cholesterol level of greater than 200 mg% the females had a higher mean BMI than the males in the 30 - 39 age group, whereas with a cholesterol level of less than 200 mg% the mean BMI of females was only greater than males in the 50 - 60 age group.

In both sexes mean systolic blood pressure increased with age and plasma cholesterol level. Males had a higher mean systolic blood pressure than females except where the plasma cholesterol was greater than 220 mg% when the mean systolic blood pressure tended to equalize. In both sexes the mean diastolic blood pressure tended to increase with age and plasma cholesterol level. Males generally had higher means.

In both sexes mean fasting blood glucose increased with age and plasma cholesterol level. Females with higher mean plasma cholesterol levels tended to have higher fasting blood glucose levels than males.

HYPERTENSION

The prevalence of hypertension was higher amongst males than females and rates rose with age for both sexes (Table 59).

There was a statistically significant association between IHD and blood pressure levels in both

sexes (Tables 60 - 63), and a steep increase in the prevalence of IHD with increase in blood pressure levels.

The highest prevalence rates were found among the hypertensives and these were 2 - 3 times as high as among the borderline hypertensives. Similarly the lowest rates were found amongst the normotensives.

The means and standard deviations of coronary risk factors by blood pressure level are presented in Table 64.

The males were taller than the females in all age groups and blood pressure levels. The males were heavier than the females in all age groups and at all blood pressure levels. The mean weights in the hypertensive group were greater than the mean weights in the normotensive and borderline hypertensive groups. The mean weights were greater in the 40 - 49 age group at all blood pressure levels than both the younger and older age groups. The 40 - 49 age group at all blood pressure levels were heavier for their height than both the younger and older age groups.

The body mass index increased with higher blood pressure in the 40 - 49 age group but no such correlation was found in the younger or older age groups.

Plasma cholesterol level increased with increasing blood pressure levels in all age groups.

Hypertensive females in the two older age groups had higher plasma cholesterol levels than the males in the same group. There was no significant difference in plasma cholesterol levels between males and females in the normotensives and borderline hypertensives, at all ages.

The mean fasting blood glucose tended to increase with age at all blood pressure levels, and increased with blood pressure. However, the females tended to have a higher fasting blood glucose in all ages and at all blood pressure levels.

SMOKING

The distribution of the study population by age, sex and smoking is shown in Table 65. The overall percentage of current smokers was 46.6% for all men and 14.6% for all women. The percentages of smokers were similar in the three age groups for men, while for women the percentage rose with age. The percentage of ex-smokers was higher among the older age groups. For current smokers, the data show that 87.5% of the men and 71.6% of the women smoked more than 15 cigarettes per day (Table 66).

Tables 67 and 68 present the prevalence rates of IHD broken down by smoking habits. The data indicate that the prevalence rate of IHD in both sexes was higher

amongst ex-smokers and smokers than amongst those who had never smoked. The high prevalence amongst ex-smokers perhaps reflects the effect of medical advice on surviving subjects.

Table 69 presents the means and standard deviations of physique and biochemical attributes. Smokers and ex-smokers tended to be taller than those who had never smoked. In both sexes the non-smokers and ex-smokers were heavier than the current smokers. The body mass index showed a similar trend.

Table 70 presents the smoking habits of patients with intermittent claudication as determined by the questionnaire. The data show a strong association between smoking and this disease in both sexes. Only one of the 26 patients with intermittent claudication had never smoked.

PHYSICAL ACTIVITY

The questionnaire on exercise in leisure time was administered by the interviewers on the basis of the Instruction Manual II VE (Morris and Chave, 1973).

The activities classified as vigorous exercise (Table 71) are those which produce peaks of energy expenditure enough to achieve conditioning and perhaps produce protective physiological changes from IHD (Chave et al., 1978).

The distribution of the study population by age, sex and exercise is presented in Table 72. The data show that about one-third of all the mean (35.9%) and one-third of all the women (37.0%) took vigorous exercise in their leisure time.

The percentages of the men who took exercise daily and weekly were higher than those for women. About two-thirds (64.6%) of the women who took exercise did so occasionally (Table 73).

The data indicate that there is significant inverse association between leisure-time exercise and prevalence of IHD (Tables 74 and 75). The association is consistent in all IHD classes.

The age-specific means and standard deviations of coronary risk factors by vigorous exercise and sex are presented in Table 76. The age-adjusted percentages of these factors are presented in Table 77.

In both sexes the age-specific means of height showed slight differences, while the age-adjust percentages of height showed that those doing exercise were shorter than those not doing exercise.

In both sexes the inactive subjects were heavier for their height than the active, and a higher prevalence of hypertension and borderline hypertension was observed in the inactive than in the active subjects.

In both sexes the means of plasma cholesterol concentration and the percentages $\geq$ 220 mg% were higher among the inactive subjects than the active.

Prevalence of hyperglycaemia and a means of fasting blood glucose were higher in the inactive subjects in both sexes.

The percentages of current smokers was higher in the inactive subjects in both sexes. Ex-smokers were more frequent in active than inactive men while in women the trend was reversed.

In both sexes, type A behaviour was more prevalent in those who did exercise.

The active subjects in both sexes ate less white bread and more brown bread.

BEHAVIOUR PATTERN

The responses to the ten-item Framingham type A questionnaire are shown in Tables 78 - 87.

In both sexes, over half of the participants were either 'very' or 'fairly well' "bossy", while more than half also ate too quickly. Over two-thirds were ambitious, competitive and would get upset when they had to wait.

The females felt more pressed for time than the males, probably because the working women had family and domestic responsibilities.

More than 40% of the men and women were stretched by work to the very limits of their capacity and often thought about work after duty hours.

Only about a quarter of the men and women felt uncertain or dissatisfied about the performance of their work.

To assess the behaviour pattern the Framingham scoring system was used. The response to each question was valued from zero to one, one indicating complete presence of the trait and zero its absence (Table 88).

The medians of the scores for each of males and females were determined and used to discriminate type A from type B individuals. Framingham type As were defined as persons who scored in the upper 50% and type Bs were defined as those who scored in the lower 50% of the scores. The greater than 50% prevalence of type A among females was due to the clustering of some psychosocial scores around the median.

The age-specific and age-adjusted prevalence rates of IHD and behaviour patterns are presented in Tables 89 - 92. There were striking differences between the prevalence of IHD in the two types of behaviour in both sexes. There was a statistically significant correlation between type A and IHD in both sexes.

Tables 93 and 94 compare type A men and women with type B in relation to the risk factors which were measured.

The type A men were heavier for their height than the type B. In women the difference was reversed.

In both sexes a higher prevalence of hypertension and broderline hypertension was observed in type A than in type B.

Also type A in both sexes had a higher percentage of plasma cholesterol ≥ 220 mg% than the other type.

Prevalence of hyperglycaemia was higher in type A men than type B. But in women the difference was the other way round.

The percentage of current smokers was higher in type A in both sexes. Ex-smokers were more frequent in type A men than type B. In women ex-smokers were roughly equally represented in both types.

In both sexes the percentage of those who did exercise in leisure time was higher in type A than in type B.

OCCUPATION

The distribution of the study population by age and sex and occupation is presented in Table 95. The data show that in men there were 4 - 5% more teachers than either administrative or manual staff, while in women there were 13 - 14% more teachers.

Over half of the participants in both sexes in the lower age group were teachers. In the upper age groups there were more manual workers and administrative staff than teachers.

The age-specific and age-adjusted prevalence rates of IHD by sex and occupation are presented in Table 96 - 99. The data show that the highest rates of

IHD were among the administrative staff whilst the lowest rates were found in the teaching profession.

In both sexes the age-adjusted angina rates for both administrative and manual workers were higher than among teaching staff.

In males the highest age-adjusted rates of PMI were among administrative staff. There was no PMI in female teaching staff; the rates of PMI amongst females who were administrative and manual workers were close.

In males, the age-adjusted rates were highest amongst administrative staff, intermediate amongst manual workers and lowest amongst teachers. In females the highest rates were amongst administrative staff, the lowest amongst manual workers and the intermediate amongst teachers.

The age-adjusted means, percentages and standard errors of means of coronary risk factors are presented in Table 100.

Among males, manual workers were shorter and administrative staff taller, teaching staff were intermediate. In females the pattern of height was reversed with manual workers the tallest.

In males, manual workers were heavier for their height than the teaching staff, but administrative workers were heaviest for their heights. In females, the manual workers were the heaviest for their height while the teaching staff were lightest for their height,

female administrative staff being intermediate.

In males the percentage of those who had systolic blood pressure ≥ 160 mm Hg was lower among teachers than both administrative and manual workers. The administrative staff had a higher mean systolic blood pressure than the manual workers but the percentage of hypertensives was very close. The diastolic blood pressure showed a similar trend.

The percentage of those who had systolic blood pressure ≥ 160 mm Hg was highest among female administrative staff and lowest among teaching staff. The diastolic blood pressure showed a similar trend.

In males the plasma cholesterol level was highest in administrative staff and lowest in manual workers. In females it was highest among administrative staff but lowest among teachers.

In both sexes there were more hyperglycaemics in manual workers and administrative staff than in the teaching profession.

The highest percentage of smokers was found among male manual workers and female administrative staff. The teaching profession smoked least.

In both sexes teachers took most exercise in their leisure time while manual workers were the least active.

HYPERGLYCAEMIA

Blood glucose was measured after fasting from the evening meal before the day of examination.

The cut-off point of 120 mg/100 ml was used and those with a level equal or higher were classified as hyperglycaemics.

It can be seen from Table 101 that the prevalence of hyperglycaemia is almost equal in men (4.4%) and women (4.5%).

The prevalence showed significant increase with age. The rates were similar for both sexes in the third decade, higher for females in the fourth decade and higher for males in the fifth decade.

The relationship of IHD to hyperglycaemia is shown in Tables 102 and 103. A significantly high prevalence of IHD was observed among hyperglycaemics in comparison to normoglycaemics.

It is interesting to note that in both sexes the prevalence of PMI ($p < 0.001$) and ischaemic-type ECG changes ($p < 0.01$) showed more significant association with hyperglycaemia than did angina ($p < 0.05$).

OTHER RISK FACTORS

Water Hardness

The water used for drinking and cooking purposes is supplied to Amman from two sources - artesian wells and springs.

The analysis of the water from the two sources was done in Amman and later in the London School of Hygiene and Tropical Medicine (Table 104).

Apart from the magnesium content there was little or no difference in the chemical composition of the water from both sources, particularly its calcium content, therefore, no analysis was done on the relationship of IHD with soft or hard water, because of very little variation in water content.

Oral Contraceptives

Women were classified on the basis of their answers to the questionnaire on oral contraceptives as users, ex-users and never-used.

About two-thirds of all the women never used the Pill. The current users were only 8.7%.

The age-specific and age-adjusted prevalence rates of IHD and oral contraceptives are presented in Table 105. In the age group 30 - 39 Pill users had a

higher prevalence of possible myocardial infarction and ischaemic-type ECG changes than other women. By contrast, in the age group 50 - 60 ex-users had a higher prevalence of angina, possible myocardial infarction and ischaemic-type ECG changes than other women. In the age group 40 - 49, pill users had a higher prevalence of angina and ischaemic-type ECG changes while ex-users had a higher prevalence of possible myocardial infarction than other women.

Alcohol

The data on alcohol consumption and its association with IHD are presented in Tables 106 - 109.

There were very few men who were current alcohol drinkers (9.8%) and even fewer women (4.9%). Also the percentage of those who had given up alcohol was low in both men (9.8%) and women (2.6%).

The prevalence of angina and ischaemic-type ECG changes was higher among drinkers than ex-drinkers and those who have never drunk. In the case of PMI the highest prevalence was among the ex-drinkers.

MULTIVARIATE ANALYSES

The results of multiple logistic regression of the prevalence of IHD are presented in Table 110 for

males and in Table 111 for females. The numbers of IHD cases were small, therefore interpretation of results should be made with caution. No inferences should be made in females on PMI and ischaemic-type ECG changes without symptoms because the numbers involved were very small.

In males, the data show that age was an independent contributor to the prevalence of ischaemic-type ECG changes and ischaemic-type ECG changes without symptoms, but its contribution to other IHD forms was insignificant.

BMI and hyperglycaemia were not independent risk factors for IHD prevalence in both sexes. Hypertension was a significant contributor to the prevalence of angina, ischaemic-type ECG changes in both sexes and also to ischaemic-type ECG changes without symptoms in males.

Plasma total cholesterol was a significant contributor to the prevalence of all forms of IHD in males and to the prevalence of ischaemic-type ECG changes in females.

Cigarette smoking contributed significantly to the prevalence of ischaemic-type ECG changes in both sexes and to angina in females.

Type A behaviour pattern contributed significantly to PMI in males and to ischaemic-type ECG changes in females.

Physical inactivity contributed to all forms of IHD in males and to ischaemic-type ECG changes in females.

Brown bread showed a significant negative correlation with ischaemic-type ECG changes in males.

Generally, these findings confirm those of the univariate analysis, however hyperglycaemia was an exception.

CHAPTER VI

DISCUSSION

LIMITATIONS AND BIAS

Prevalence rates are less telling than incidence rates because they necessarily apply only to survivors and because there are greater difficulties in drawing aetiological inferences. However, prevalence studies are easier, covering as they do a shorter space of time and yield valuable data as long as their limitations are kept in mind (Epstein, 1965).

The interpretation of results from cross-sectional studies is often fraught with difficulties, including the inability to determine antecedent-consequent relationship (Haynes et al., 1978).

It has been shown that the cultural background of the respondents and the mode of administration of the questionnaire can affect prevalence estimates.

In view of these limitations, it is necessary to examine the factors which may have influenced the findings of this study. The factors are:

1. Non-Response

Non-response was low (8.6%) for the total study

population. A check of the medical records of the non-respondents showed that they were generally representative of the whole study population and were not drawn from a single age, sex or occupational category. It seems unlikely that the non-response could have had a major effect on the study results. Checking of the medical records of the non-respondents revealed that none of them had any IHD.

2. Observer Bias

The subjects were randomly allocated to the two interviewers. The methods used were standardized to remove as much interviewer bias as possible. The interviewers were adequately trained before the study started and their results were checked at irregular intervals of 4 - 6 weeks and were found to be consistent. The scales used for measurement of weight and height and sphygnomanometers were calibrated before and during the study.

The questionnaire results and the means and standard deviations of the data collected by the two interviewers were close.

There is no reason to believe that the two interviewers compared their results or consciously misrepresented any recording.

3. The Questionnaire

There are problems in translating and applying a questionnaire in a different culture with respect to validity and comparability between and within studies. However, attention was paid to proper translation, testing before the study started and making within observer and inter-observer comparisons during the study.

There was a corroboration between the questionnaire results and ECG findings. The IHD rates also showed age-gradient and sex ratio. These results are consistent with expectations external to the study.

All these factors suggest that the questionnaire bias was of little or no account.

4. Misclassification

The criteria used for diagnosis of IHD and ECG coding were clearly defined and strictly applied. Nevertheless, it is difficult to rule out any misclassification and still more difficult to determine its extent.

In studies which do not utilize formal clinical examination in their methods, it may be more difficult to exclude subjects with rheumatic, congenital or other heart disease.

5. Antecedent-Consequent Variations

It is likely that the characteristics of some participants may have changed subsequent to development of IHD or as a result of medical advice, for instance they may have altered their smoking, diet, physical activity and behaviour pattern.

It is also likely that the subjects who had the highest levels of risk factors (such as severe hypertension) were removed from the population at risk by disability or death leading to low means of these risk factors in those remaining in the study.

Also the levels of risk factors in some cases may have been reduced by being under medical treatment for related conditions.

Bearing the above points in mind, the important question to arise is whether these changes occurred in a sufficient number of the participants to influence the associations observed.

In a cross-sectional study it is difficult to find a precise answer to this question. However such problems may be reduced in a longitudinal study.

Despite the above limitations, there is evidence to suggest that the associations found may have not been appreciably influenced by the antecedent-consequent relationships of IHD.

1. The demonstrated associations between ischaemic-type ECG changes and coronary risk factors still holds

true when ECG cases with positive symptomatic history were excluded from the analysis.

2. The higher prevalence of IHD in ex-smokers suggests that people may have given up smoking after developing the disease. Despite this the prevalence of IHD among current smokers was still higher than among those who have never smoked.

3. All the participants were interviewed while leading their ordinary lives. None was interviewed or examined during the illness or the convalescent period.

4. It has been shown that plasma cholesterol reverts to the pre-infarction levels about three months after infarction (Tibblin and Cramer, 1963; Cotton, 1970; Fyfe et al., 1971).

5. Jenkins et al. (1976) have shown that mean type A scores do not differ significantly as a function of whether coronary events occurred before or after taking the Jenkins Activity Scale.

6. Prospective analyses by Rosenman et al. (1975) showed that type As with IHD were equally as likely at the eight and a half year follow up to die from their coronary disease as were type Bs with coronary disease. It seems unlikely therefore that type As with IHD would be selectively removed from the population at a greater rate than type Bs with IHD.

In conclusion it does not seem likely that all the observed relationships were the result of methodological

problems. However, the study was cross-sectional and the limitations of such studies should be borne in mind when the results are interpreted.

AGE AND IHD

The age-specific and age-adjusted prevalence rates of IHD for both sexes in relation to the coronary risk factors have been calculated and presented individually.

In both sexes in univariate analysis the prevalence of IHD increased significantly with age. However, a lack of age-IHD relationship has been noted in certain instances. In both sexes the prevalence of PMI among hypertensives diminished with increasing age, and in females, the prevalence of angina diminished with age when the plasma cholesterol level was above 240 mg%. In a cross-sectional study these irregularities are to be expected and the likely causes have been discussed earlier.

It is clear that there is an age trend in the prevalence of IHD in this cohort. The question which may arise is whether this trend is due to the time-dose product of acquired risk factors or simply due to the biological effect of ageing.

There is no easy answer to this question; it is probable that both the dosage of the coronary risk factors and the altered tissue response are involved. However, the effect of age on two risk factors, namely

cholesterol and hypertension, merits consideration.

The rise in serum cholesterol level with age observed in this and other studies, varied among different populations. Keys (1970) and Stamler (1974) attribute these variations to differences among populations in regard to habitual diet, particularly saturated fat and cholesterol intake.

With regard to blood pressure, Miall et al. (1968) have shown that age appears to have very little influence on blood pressure, after the effect of other variables, particularly increases in body weight, has been removed.

Miall (1971) later suggested that blood pressure itself, rather than age, determines its subsequent rate of increase, possibly by precipitating changes in the vascular bed.

In contrast, it has been argued that there are probably some pathological changes attributable to ageing which may contribute towards the increase in blood pressure with age. It has been noted that the atherosclerotic process contributes to loss of vascular elasticity and leads to an increase in systolic pressure levels (Goldstein, 1971; Cawley and Guefton, 1975).

IHD PREVALENCE RATES

The results of this survey are a cross-sectional view of IHD and its attributes as they present themselves

in the UNRWA workforce in Jordan. The age-specific and age-adjusted prevalence rates of angina and possible myocardial infarction are high. These rates were paralleled by similar rates of ischaemic-type ECG findings.

International comparisons of IHD prevalence rates are confounded by differences in population sampling, methods and diagnostic criteria. Therefore any comparisons between prevalence rates reported by other studies with these findings should be interpreted with caution. However, and with regard to the above reservation, the IHD prevalence rates of this cohort approximate to those of the Tecumseh study (Epstein et al., 1965) and the Busselton study (Welborn et al., 1969) and the Chandigarh study (Sarvotham and Berry, 1968). The rates for males also approximate to those reported in the prevalence comparisons in Europe (Rose et al., 1968).

As this study is the first to be conducted in Jordan on the prevalence of IHD, there are no available data for this country with which its findings may be compared. Also, reliable data on IHD incidence, mortality and case-fatality in Jordan are not available. Therefore it was not possible to study the relationship between the prevalence rates observed and these parameters. However, the number of Jordanians who underwent aorto-coronary by-pass operations at the King Hussein Medical Centre in Amman during the year 1979 are presented in

Table 112. Although these figures are an underestimate, because they do not include those Jordanians who were operated upon abroad, nevertheless they affirm how prevalent this disease is in Jordan.

The question which arises now is, why is there a high prevalence of IHD in this cohort? In answer to this question we should take a rapid glance at the changes which preceded and accompanied the rise in the prevalence of this disease in developed countries in the 1950s and 1960s. It has been suggested that most of the burden of IHD in these countries was the result of modern technology and affluence which became predominant at the time. These have led inter alia to a greater use of non-human energy in mass-production and everyday life, the disappearance of physically strenuous occupations, mass consumption of cigarettes, intake of rich diets and conflicts of modern life (pace, mobility, competitiveness, etc.) with their associated changes in behavioural patterns. The drive amongst Jordanians to become developed has resulted in their acquisition of the positive as well as the negative characteristics of the affluent societies. Among the negative facets have been the prerequisites for the prevalence of IHD.

Further, in Jordan, the availability of modern cardiovascular facilities helped, not only to establish accurate diagnosis of the disease, but also to detect it in milder forms.

ISCHAEMIC-TYPE ECG CHANGES

The prevalence of ECG changes shows an age gradient and sex ratio which parallel the prevalence of IHD as determined by the standard questionnaire. The finding that about 33% of angina-positive are ECG-positive is in line with that reported by Rose et al. (1968). Although angina and ischaemic-type ECG changes are different categories of IHD, this contrast lends support to the validity of the questionnaire application and ECG codings made in Jordan.

Comparison with Other Studies

Table 113 shows comparisons for ischaemic-type ECG changes between the males of this study aged 40 - 49 and those from Lawrence Tavern (Miall et al., 1972), Framingham (Higgins et al., 1965), Tecumseh (Ostrander, 1966) and four European studies (Rose et al., 1968). The rates for Q/QS changes are similar to those reported by these studies. However, the rates for left-axis deviation and high-amplitude R waves are lower in this study than in the other studies. The rates for ST depression are lower than the Jamaican and American rates but they approximate to the European rates. The rates for T wave inversion and flattening are lower than in all the other studies. The rates for the left

bundle branch block are similar to those reported by other studies.

Despite the limitations of inter-survey comparisons the ischaemic-type ECG changes in this cohort appear to approximate to those in Western populations.

Ischaemic-Type ECG Changes without Symptoms

Over half of those diagnosed as having ischaemic-type ECG changes had no history of angina or possible myocardial infarction. This suggests that a high proportion of these ischaemic changes did not become clinically manifest or it may be that some people have a defective anginal warning system.

It is interesting to note that a statistically significant correlation was found between ischaemic-type ECG changes without symptoms and coronary risk factors such as physical exercise. This suggests that the correlation between these risk factors and IHD is true and is not the result of a change of habit after developing the disease.

Comparison with Other Studies

Research on ischaemic-type ECG changes without symptoms has not been encountered. Asymptomatic myocardial ischaemia has been studied. The two most notable studies in this field were those of Erickseen et al. (1976) and of Froelicher et al. (1976). The former

group showed that 3.5% of more than 2,000 men aged 40 - 59 had coronary artery disease. In the latter group of 1,390 asymptomatic Air Force crewmen aged 35 - 55 the rate was 2.2%. These studies would suggest that the prevalence of silent ischaemia is between 2 and 4 per cent.

Although asymptomatic myocardial ischaemia and asymptomatic ECG changes may not be the same, they are possibly related, but further research in this area is still required.

Whether the silent cardiac ischaemia involves less myocardium than angina-related ischaemia is not clear. Nor is it clear if the severity of coronary artery disease determines the occurrence of these silent ischaemic changes. In such people ischaemic changes presumably go unnoticed until a complication occurs or the change is detected in a routine electrocardiogram. Can one assume that it is in these subjects that many of the sudden deaths will occur?

DIET AND IHD

The relationship of diet with IHD was examined in three categories of food consumption - with all food items, with fats only and with bread intake. The scoring and analyses were based on the hypothesis that certain food items would be protective against IHD whilst the other might be detrimental.

There was no significant correlation between either the dietary protective factor or fat protective factor and the prevalence of angina and PMI. There were, however, statistically significant correlations between the dietary protective factor, the fat protective factor and the prevalence of ischaemic-type ECG changes without symptoms. A significant correlation was also found between fat protective factor and ischaemic-type ECG changes. Furthermore, there was a significant correlation between the bread factor and the prevalence of angina, PMI and ischaemic-type ECG changes.

In multivariate analysis the correlation between the bread factor and the prevalence of ischaemic-type ECG changes in males remained significant after controlling for other coronary risk factors.

The data also revealed some interesting but not statistically significant correlations. Both men and women who ate vegetable rather than animal fats, brown rather than white bread, more beans and fruit and less meat, had less angina cases. But there was little difference for cases who had possible myocardial infarction, possibly due to a change of habit because some of them knew that they had the disease or had been given dietary advice.

That most epidemiological studies carried out within homogeneous populations have failed to demonstrate a significant correlation between diet and IHD is well

recognized in the literature (Paul et al., 1963; Morris et al., 1963; National Diet-Heart Study, 1968; Khan et al., 1969; Keys, 1970; Nichol et al., 1977). Nevertheless, a significant correlation between bread and IHD risk has been reported by a few investigators (Morris et al., 1963, 1978; Trowell, 1972).

Whilst it may be argued that the questionnaire used for the collection of dietary data was not an entirely reliable method of assessment, the use of alternative methods was not possible due to limited time and resources. There are however, a number of positive reasons for believing that the limitations of the method were not responsible for appreciably reducing the relationship between diet and IHD. The participants were of an age range in which the dietary habits tend to be consistent and stable. Furthermore, in Jordan, in contrast to Western societies, the husbands do the shopping, while the wives prepare the food. These two factors helped in the collection of a reliable long-term dietary pattern and minimised errors in the reporting of dietary habits. In addition, the participants had diets that differed from each other's and the range of their nutrient intake seems to be wider than the range in western societies, but not sufficiently wide enough to demonstrate convincingly the correlation between dietary items and the prevalence of IHD.

On the other hand, there may well be other

endogenous and exogenous factors which may have masked the correlation between diet and IHD. It is possible that dietary factors may be working in opposition, cancelling out each other's effect, and it is also possible that non-dietary factors which raise or lower serum total cholesterol may have countered the dietary effect.

Diet and Coronary Risk Factors

Those who ate detrimental diets tended to be heavier than those who ate protective diets and a higher proportion had a body mass index $\geq$ 28. Differences in dietary intake might account for these differences in physique. Differences in energy expenditure might also be part of the explanation. The age-adjusted percentages of subjects who did physical exercise in leisure time were higher among those who ate protective diets than those who ate detrimental diets.

In both sexes the age-adjusted percentages of hypertensives and broderline hypertensives were higher among those who ate detrimental diets, than in those who ate protective diets. The means of both systolic and diastolic blood pressures showed a similar trend. The associations between diet, weight and hypertension are in agreement with previous observations (Kannel et al., 1967; Johnson et al., 1973; Oberman et al., 1967; Miall et al., 1968; Paffenbarger et al., 1968; Johnson et al., 1973; Ulrych et al., 1973).

It is interesting to note that in both sexes the age-adjusted percentages of those who had cholesterol levels ≥ 220 mg% were higher among those who ate a detrimental diet. Mean serum cholesterol showed a similar trend.

In women the means of fasting blood sugar and age-adjusted percentages of fasting blood sugar ≥ 120 mg% were higher among those who ate a detrimental diet while in the males the findings were inconclusive. It is probable that dietary advice given to diabetic patients has influenced these results.

In both sexes the age-adjusted percentages of current smokers were higher in those who ate detrimental diets. This finding leads to the conclusion that those who ate protective diets are more health-conscious than the others as they also exercised more. The association between behaviour pattern and diet intake was intriguing. Whilst in men type A behaviour pattern was exhibited more among those who ate detrimental diets, in women it was exhibited more among those who ate protective diets. The reason for this variation in behaviour between the two sexes is not clear.

The associations between fat intake and other coronary risk factors were similar to those found between the dietary protective factors and these factors. This is partly due to the inclusion of the scores for fat consumption in the dietary protective factor. The

associations between bread intake and coronary risk factors were interesting. In both sexes those who ate white bread were found to be heavier than those who ate brown bread alone or brown and some white. Also in both sexes there were more hypertensives amongst those who ate white bread. In both sexes the age-adjusted percentages of those who had serum total cholesterol $\geq$220 mg% were highest in those who ate white bread.

The age-adjusted percentage of males and females taking exercise in leisure time was highest in the group who ate the brown bread, lowest in the white bread eaters and intermediate in those who ate both. Possibly this reflects the fact that the more health-conscious people both eat brown bread and take exercise.

With regard to type A behaviour pattern for males the age-adjusted percentage was highest for males among brown bread eaters, intermediate among the white bread eaters, and lowest among those who ate both types. However, for females the highest percentage was in the white bread group, the intermediate in the group eating both types and the lowest among those who ate brown bread.

In conclusion the correlations observed between diet and other coronary risk factors are in line with the correlations observed between diet and IHD.

PLASMA CHOLESTEROL

In males the means were higher than in females for the younger and older age groups, while for the intermediate age group the trend was reversed. In both sexes the means increased with age.

Slack et al. (1977) reported on serum total cholesterol concentration of north-west London residents, and Heiss et al. (1980) reported on the cholesterol concentration of North American Lipid Research Clinic's participants. The age trends by sex of plasma total cholesterol reported by this study are similar to those presented by Slack et al. and Heiss et al.

An important consideration when means of total cholesterol are compared is the problem of the variation in methods used in different studies. Under the best conditions the standard error of measurement in blind duplicate analyses of serum total cholesterol by either the bench Abell-Kendall method, or the automatic "auto-technicon" method, is about $\pm$ 2% of the mean value (Keys, 1969). Of course, under less than ideal conditions, or with the other methods, this source of variation may be greater. Furthermore, the Abell-Kendall method gives figures about 15% lower for total serum cholesterol concentrations than the direct methods (Schwartz and Hill, 1972).

Thus, the studies comparable to this one, with regard to serum total cholesterol values, are those which used the Abell-Kendall method or were adjusted to that method. Although this selection criterion may help to reduce some of the analytic variation, it is much more difficult to control for other methodological details that may influence the cholesterol results. These include age, sex, ethnicity, blood-sample collection methods, occurrence of haemolysis, storage and transportation of blood samples, and whether cholesterol is estimated on serum or plasma (Beaglehole, 1979). Plasma cholesterol estimations are about 3% lower than serum levels (Laboratory Methods Committee of Lipid Research Clinics Programme, 1977).

It has been shown that as cholesterol concentration increases, IHD risk also increases, in other words the relationship is continuous. There is little evidence of a threshold between normal and abnormal. Despite this an arbitrary choice of cut-off values for the upper limits of total cholesterol distribution was used to estimate its frequency in a community. Thus with a cut-off point of 240 mg%, the overall prevalence of raised total cholesterol in this study was 17.2% for males and 10.3% for females. In a study from California, Wood et al. (1972) used similar cut-off points and found prevalence rates of 19% for males and 10.9% for females. Although the subjects and methods in these studies were

not directly comparable they are helpful in suggesting international trends. Furthermore, it should be noted that any serum total cholesterol value suggested as an upper limit will have different implications, for the proportions of men and women exceeding the limit at different ages, and for individuals seeking advice about hypercholesterolaemia.

IHD and Plasma Cholesterol

There was significant association between IHD and plasma cholesterol level. There were also significant differences in the prevalence of IHD with age and sex. There were no significant interactions between age and cholesterol, and sex and cholesterol. When adjustments are made for age and sex, the association of IHD with cholesterol level remained highly significant. Also, multivariate analysis showed that plasma cholesterol level was still a significant factor in IHD prevalence after controlling for other coronary risk factors. These findings are in line with epidemiological evidence from within different cultures where the relationship between serum cholesterol and IHD was examined by cross-cultural comparisons.

Plasma Cholesterol Level and Coronary Risk Factors

In males weight and body mass index rose with increase in plasma cholesterol

level. A similar trend was found in females. In both sexes means of systolic blood pressure rose with the increase in plasma cholesterol level while means of diastolic blood pressure tended to do the same. Also in both sexes means of fasting blood sugar increased with plasma cholesterol level. These findings are in agreement with what has been reported in literature concerning the correlation between cholesterol levels and these risk factors.

HYPERTENSION

Intra-individual variability of blood pressure readings and between- and within-observer errors of blood pressure measurement are widely recognized as sources of methodological bias in the epidemiological studies of hypertension.

Armitage and Rose (1966) conducted studies on the variability observable within and between occasions of observation and demonstrated wide differences both individually in the degree of variability and greater between than within occasion variation among their subjects. Also, Evans and Rose (1971) identified several factors accounting for systematic errors in blood pressure measurements, such as circumstances of measurement,

physical characteristics of the arm in relation to dimensions of the cuff, seasonal changes and between-observer differences.

Furthermore, blood pressure tends to be higher on initial measurement because of what Pickering (1972) termed the "investigatory" or "defence reflex" due to excitation involving the cardiovascular system resulting from exposure to a potentially threatening situation.

With these difficulties taken into consideration, the training programme which preceded this survey included orientation to the problems in standardisation and quality control of blood pressure readings among observers. The fact that the participants had previously undergone periodical medical examination which included blood pressure measurement may have removed the effect of the "investigatory" reflex. Despite these precautions it was difficult to eliminate methodological bias.

The means of systolic and diastolic blood pressures increased with age in both sexes. Also the prevalence of high blood pressure showed a similar trend. Apart from certain rural communities in which blood pressure does not rise with age, in most populations the mean blood pressure level rises progressively with increasing age in both sexes (Shaper, 1974).

The differences between the means of men's and

women's blood pressure in each age group diminish with increasing age. Thus although the females' pressures are lower than the males' pressures the ageing effect appears to be the same in both sexes.

Hypertension was more prevalent among males than females in the three age groups. Also the means of systolic and diastolic blood pressure were higher among males than females in all age groups.

There was a clear association between blood pressure level and weight. The mean weight of the hypertensive group was highest, that of the borderline hypertensives was intermediate, and that of normotensives was lowest. The present study confirms the previously reported correlation between prevalence of hypertension and weight (Kannel et al., 1967; Chiang et al., 1969; Stamler et al., 1975).

Thus, it is plausible to hypothesize that, as people become older, elasticity in longer arteries wanes, the resistance in smaller vessels increases and thus any concomitant excess in weight will aggravate the tendency to hypertension.

Freis (1976) and Page (1976) have reviewed the epidemiological evidence on the aetiology of human hypertension. Both have underlined the importance of salt intake and body stature on the prevalence of hypertension, its rise with age and its influence on the differences observed in acculturated populations. It is therefore likely that a better understanding of the

dietary and other environmental characteristics which distinguish populations whose pressures rise with age from those whose pressures rarely do so (Pickering, 1968; Prior et al., 1968; Truswell et al., 1972; Jorgenson et al., 1972; Oliver et al., 1975) will clarify the aetiology of hypertension.

IHD and Hypertension

In both univariate and multivariate analyses there was a significant correlation between IHD and hypertension. The prevalence showed a gradient with the lowest rates among the normotensives, the intermediate among the borderline hypertensives and the highest among the hypertensives. This rise in IHD prevalence with increase in blood pressure level argues against the existence of any critical dividing line separating the normotensives from those at higher risk.

CIGARETTE SMOKING

There are two sources of supply of cigarettes in Jordan. Some are manufactured in three large factories in Amman and the rest are imported from all over the world. Data on annual cigarette consumption per capita were not available and data on local production and sales were withheld by the manufacturers.

Of the population studied, those who reported

that they were current smokers were 46.6% of all males and 14.6% of all females. Most of those who smoked did so heavily. It is likely that women under-reported their smoking habits particularly the light smokers. This may be attributable to reluctance among Arab women to discuss personal practices.

Smoking and IHD

There were highly significant differences in the prevalence of IHD with age, sex and smoking habits. In multivariate analysis the correlation between cigarette smoking and ischaemic-type ECGs in males, and angina and ischaemic-type ECGs in females remained significant when the other risk factors were controlled. In males the prevalence of IHD was higher in ex-smokers than in smokers or those who had never smoked. It may be that many ex-smokers had stopped on becoming aware that they had IHD. In contrast, in females, the prevalence of IHD was higher among smokers than those who had never smoked.

These findings are in line with those from several previous studies, which have established smoking as a major risk factor for IHD (Doyle et al., 1962; Hammond and Garfinkel, 1969; Rogot, 1974; Doll and Peto, 1976; The Pooling Project Research Group, 1978).

Smoking and Coronary Risk Factors

In both sexes, smokers and ex-smokers tended to be taller than those who had never smoked. Similar

trends in height differences have been observed in other studies (Seltzer, 1963; Higgins and Kjelsberg, 1967; Manson, 1970).

In both sexes, current cigarette smokers were of a lower average weight than ex-smokers and those who had never smoked. The body mass index showed a similar trend. These findings are also consistent with previous studies (Bjelke, 1971; Khosla and Lowe, 1971, 1972, 1973; Kopczynski, 1972; Hawthorne et al., 1979).

In males there was a higher cholesterol level in current cigarette smokers than in ex-smokers and those who had never smoked. This differed from the female group where higher cholesterol levels were found in ex-smokers and smokers than in those who had never smoked. Heavy cigarette smokers have been shown to have higher average serum cholesterol values than those who have never smoked in previous studies (Thomas, 1960; Pincherle, 1971; Dales et al., 1974; Holme et al., 1976).

It has been suggested that raised serum lipid levels in smokers may be a result of dietary differences because food preferences differ according to smoking habits (Bronte-Stewart et al., 1960). The lower cholesterol value in those who had never smoked cigarettes may be due to health-conscious individuals in this group who had probably changed their dietary and physical activity habits compared with heavy cigarette smokers.

However, overeating among cigarette smokers compared with those who had never smoked cigarettes was

not the case, as the mean body weight in smokers was lower than in those who had never smoked cigarettes, confirming previous observations (Khosla and Lowe, 1973). Nevertheless a qualitative difference in the diet pattern between cigarette smokers and those who have never smoked cigarettes is, of course, possible.

It has been shown that differences in metabolism exist between smokers and non-smokers. An increase in body weight and a decrease in oxygen consumption, heart rate and protein-bound iodine level in blood have been shown following giving up smoking (Batterman, 1955; Glauser et al., 1970). If the opposite changes took place in cigarette smokers one would expect a lower body weight in cigarette smokers than in those who had never smoked cigarettes. Thus the lower body weight of cigarette smokers may be partly due to metabolic changes causing weight reduction.

An immediate and highly significant increase in cholesterol levels lasting for 90 minutes in most of the test subjects after a single cigarette has been reported by Devi et al. (1975).

In males, the systolic blood pressure tended to be lower among smokers than both ex-smokers and those who had never smoked, whereas for the diastolic blood pressure, the trend was reversed. The literature on the association between smoking and blood pressure is divided. Some studies have reported a positive correlation (USPHS:

The Health Consequences of Smoking, 1971; Larson and Silvette, 1971; Seltzer, 1974; Kesteloot and Van Houte, 1974), while others have reported a negative correlation (Miall and Oldham, 1958; Dawber et al., 1959; Jenkins et al., 1968; Leren et al., 1975; Gordon et al., 1975).

Serum cholesterol was higher among ex-smokers than those who had never smoked. This finding is in line with previous reports (Pincherle, 1971; Gordon et al., 1975). It is likely that earlier cigarette smoking had caused persistent metabolic changes or that dietary habits had not changed after stopping smoking.

With regard to weight, the tendency of ex-smokers to gain weight after stopping should certainly not be used as an excuse for restarting. The hazards of cigarette smoking outweigh the uncertain dangers of being overweight (Mann, 1974; the Pooling Project Research Group, 1978).

EXERCISE IN LEISURE TIME

Office work generally requires a low level of physical activity. Also modern mechanisation and increasing use of cars, T.V. and food storage facilities indicate that people who are being urbanised tend to lead sedentary lives. This is why the exercise which any person can do in his leisure time has become important.

Notable growth in leisure time physical activity and participation in sports of all kinds has taken place in

Jordan in recent years. Overall, 35.9% of the male and 37.0% of the female study participants stated that they took exercise in leisure time. With the growing interest and participation in sports one would hope that the Jordanian population would become slimmer and the incidence of IHD would decline.

Two hypotheses have been postulated to relate physical activity to IHD. The first is about total activity expenditure, calorific balance and muscular development, the second about high levels of energy output, the improvement of cardiovascular efficiency (the ability to take up oxygen and the response of the heart rate to standard exercise), and the metabolic effects of exercise.

However, as a result of the reduction in occupational activity and as strenuous exertion is more likely to have declined with modern mechanisation, and as only weak associations have been found between relative weight and IHD, and because no association could be found between estimates of total physical activity and incidence of IHD (Morris et al., 1973; Morris, 1978; Chave et al., 1978), the association between high-intensity exercise and IHD is considered in this study.

The standard definition of 'heavy work' in industrial physiology is work liable to require peaks of energy expenditure of 7.5 kcals per minute or more (Astrand and Rodahl, 1970). This has been followed as

the criterion of vigorous exercise activities. These activities included active sport recreation, keep-fit exercise, heavy physical work, do-it-yourself, vigorous getting about and climbing stairs. It was postulated that all these included bursts of high-intensity 'peak' activity.

Exercise and Prevalence of IHD

In both univariate and multivariate analysis there was a significantly higher prevalence of IHD among those not doing exercise.

Overall, the age-adjusted prevalence rates of angina among inactive subjects of both sexes was 13 times that among the active. For both PMI and ischaemic-type ECG abnormalities, the age-adjusted prevalence rates among inactive males was a factor of 11 over active males. No cases of PMI were reported by the active females, whereas for inactive females an age-adjusted rate of 1.1% was found. For ischaemic-type ECG abnormalities the age-adjusted prevalence rate was 7.8 times higher among inactive females than active. For those who had ischaemic-type ECG abnormalities without symptoms, the age-adjusted rates were 5.5 times and 3.5 times higher for inactive males and females respectively than the active.

These results are in line with epidemiological studies which have suggested that high physical exercise provides a protective effect against morbidity or mortality

or both, from IHD (Morris et al., 1973; Paffenbarger and Hale, 1975; Froelicher, 1976; Chave et al., 1978; Huttunen et al., 1979).

Exercise and Coronary Risk Factors

The mechanism by which physical exercise influences coronary risk factors is not known.

Muscular activity may directly protect the cardiovascular system through neural and haemostatic mechanisms or by increasing the vascularity of myocardium.

Alternatively, physical exercise may have beneficial effects on the risk factors of IHD such as serum cholesterol and blood pressure.

In this study the means of serum cholesterol were lower in the active subjects than the inactive in both sexes and for all age groups.

In addition, the age-adjusted percentages of serum cholesterol $\geq$ 220 mg% were significantly higher amongst those not doing exercise. These results are in agreement with the studies which have shown that subjects active at work or during leisure time tend to have lower serum cholesterol concentrations than those with a sedentary occupation or life style (Hickey et al., 1975; Montoye et al., 1976; Epstein et al., 1976; Cooper et al., 1976; Huttunen et al., 1979).

Considerable evidence has accumulated indicating that the serum level of HDL is inversely related to the

development of IHD (Miller et al., 1976; Castelli et al., 1977; Gordon et al., 1977). Also the concentration of HDL has been demonstrated to be high in subjects with vigorous physical activity (Lopez et al., 1974; Wood et al., 1976; Lehtonen et al., 1978; Huttunen et al., 1979). Although this lipoprotein was not measured for the study participants, due to the unavailability of facilities in Jordan at present, the above references have been mentioned in this discussion to show the effect of exercise on an important lipid risk factor.

It is of particular interest to note that the means of both systolic and diastolic blood pressures were lower among the active than the inactive subjects in both sexes and in all age-groups - the only exceptions were in the female age group 30 - 39. Also the age-adjusted percentages of broderline hypertensive and hypertensive subjects were significantly higher among the inactive for both sexes.

Mean weight, in the various age groupings was lower among the active than the inactive subjects in both sexes. Also the age-adjusted percentages of BMI $\geq$ 28 were significantly lower among the active than the inactive in both sexes. These results are obviously understandable inasmuch as physical exercise can help in reducing weight and conditioning the body.

The means of fasting blood glucose were lower among the active than the inactive subjects in both sexes

and for all age groups. Also the age-adjusted percentages of fasting blood glucose ≥ 120 mg% were significantly lower among the active for both sexes. It is likely that the high energy output required of those taking vigorous exercise reduced glucose and fat in those subjects. It is also likely that some hyperglycaemics had transferred from the active to the inactive group for health reasons.

The active group, being more health-conscious than the inactive, smoked less and ate less white and more brown bread than the inactive.

However, it is surprising that the percentages of type A behaviour pattern were higher among the active than the inactive subjects. It appears that subjects who are proud of their socio-economic or professional achievements indulge also in recreations which are regarded as socially respectable and praiseworthy.

Whereas the lower percentages of BMI, blood pressure, serum cholesterol ≥ 220 mg%, fasting blood glucose ≥ 120 mg%, cigarette smoking and white bread intake among the active subjects may protect from IHD, the higher percentages of type A behaviour may have an opposite effect.

The question which arises here is, whether the inverse correlation between exercise and IHD is a direct result of the training or is due to accompanying changes in well-established risk factors that might be altered by physical exercise.

In multivariate analysis physical inactivity was a significant and independent contributor to IHD prevalence. Thus it may be concluded that although coronary risk factors are likely to be modified by physical exercise, the latter has a directly protective effect against IHD.

Influence of Other Factors

It is reasonable to identify the possible factors in an observed relationship and then by a process of elimination to see which non-causal factors can be excluded. Thus, the field of possible contributors can at least be narrowed and confidence in the causal hypothesis increased.

In principle, however, other explanations could account for all or part of the observed relationship between exercise and IHD. In the following discussion I shall explore sources of bias that might have contributed to the observed association between IHD and exercise.

The main non-causal explanations are physical activity shifts before or after disease, misclassification co-variables and chance effects.

1) Physical Activity Shifts

Stability of behaviour pattern: Exercise habits may have changed with the passage of time. The data reported by this study describe current exercise habits. They do not

show how many of the participants doing exercise in leisure time have continued from their youth and how many had done so but did no longer.

However, the stability of the behaviour is important for the validity of the data and their indication of physical exercise. As physical exercise is an integral part of the education curriculum in Jordan, most of the participants did exercise during their school life. During their adulthood, the special features of their employment must have affected their behaviour in their spare time. Their duty hours were from 7.30 a.m. to 2 p.m. Thus in addition to the week-ends they had ample free time in the afternoon to pursue their interests. Also, the behaviour pattern of most men and women usually becomes stable between the ages of 30 and 60.

Furthermore, as the study was conducted during a whole year, the influence of seasonal variations in doing exercise was minimised.

Physical activity shifts after disease: It may be argued that activity had changed after the onset of IHD. Subjects who became ill might have stopped doing physical exercise during leisure time. This was the likeliest alternative to a protective effect. However, the persistence of the relationship between physical exercise and ischaemic-type ECG abnormalities, when subjects with symptoms were excluded from the analysis, was still evident. Thus despite the imperfection of the methods,

the evidence seems fairly convincing that post-disease shifts in physical activity do not account for the physical activity-IHD relationship.

2) Misclassification

There are numerous forms of exercise and there is considerable variation among them. Thus it is likely that few activities that were vaguely described by the participants were misclassified by the observers.

Likewise, there are individual variations in fitness and consequently in achieving benefits of physical exercise. It may be very difficult to rule out the benefits of activities other than those arbitrarily categorised here.

Variation in the classification of moderately heavy and newly-undertaken activities was unusual. In case of doubt the classification was referred to the supervisor.

There were no missing data. The questionnaire section on physical exercise was clear. The association was consistent in all age classes. It is hard to envisage how data bias would have created the observed association.

3) Covariables

In univariate analysis statistical adjustment was made for age and sex, but the physical exercise-IHD relationship persisted. In multivariate analysis other

coronary risk factors were controlled but the physical exercise-IHD association remained significant. If the relationship was due to association between IHD and these factors, or if they were an intermediate causal link between physical exercise and IHD, adjustment would be expected to reduce the association.

4) Chance

It may be argued that the association found between physical activity and IHD could have been a chance result. However, in view of the statistical significance of the observed relationship between physical exercise and IHD, this explanation seems untenable.

Conclusion

Although not all non-causal explanations could be examined, none that was examined explained the physical exercise-IHD relationship. Therefore it may be concluded that the findings of this study are consistent with a protective hypothesis based on a direct effect of physical exercise on the cardiovascular system.

Without a more definitive controlled intervention experiment, this finding provides a basis for the possible protective effect of physical exercise against IHD.

BEHAVIOUR PATTERN

Definition

The type A behaviour pattern is a 'particular action-emotion complex' which is exhibited by an individual, who is struggling relatively excessively to obtain an inordinate number of advantages too quickly in the face of opposition or competition. This person tries to achieve more and more in less and less time, or to oppose one or more people either willingly or unavoidably.

The type A individual exhibits exaggerated personality traits of aggression, ambition and competition, is work-oriented, is often preoccupied with deadlines, and exhibits severe impatience.

The type B individual, in contrast, is mainly free of such personality traits and accordingly is free of any habitual sense of urgency or conflict.

Challenges from the Milieu

The change from a rural to an urban life style has been achieved in Amman in the last 3 decades. The people about whom the change has occurred have, as a result of the challenges imposed upon them, have had a tendency to exhibit a type A behaviour pattern in order to meet the demands of the new life.

With the increasing urbanization, technological progress and density of population in Amman, the development and civilization present challenges never experienced by earlier generations not aware of change.

Prevalence of Psychosocial Traits

1) The trait of 'being bossy or dominating'

Males described themselves as being more dominating than females in all age groups and that the younger age group considered themselves as being less dominating than both older age groups. These findings are in line with the social ranking of the population in Jordan.

2) The trait of 'having a strong need to excel in most things'

Males felt a stronger need to excel than females in all age groups when the presence of the complete trait was considered. However, when the trait was considered to apply only 'fairly well' or 'somewhat' the percentages of females exceeded those of males in all age groups.

The younger individuals were more ambitious than their elders in both sexes. This may be explained by a need for the younger population to build themselves whereas those in later life are already well established. This is particularly exhibited in those who consider the trait to apply to them 'very well'.

3) The trait of 'usually feeling pressed for time'

In the younger age group, when the full trait

was considered, the females felt more pressed for time than males. This trend was reversed in the older age groups. The exhibition of this behaviour pattern decreased with increasing age.

Of those who felt that this trait applied fairly well, the age group 40 - 49 showed a higher percentage than either the younger or older age group in both sexes. Whereas in the 'somewhat' group the trait was more prevalent in the older age group than in the younger population.

4) The trait of being 'hard driving and competitive'

The data for this trait show a similar pattern to that of being pressed for time. Presumably this is because those who are hard-driving and competitive have more to do in their time than those who show lesser degrees of the trait.

5) The trait for 'eating too quickly'

Of those exhibiting the full trait, males ate more quickly than females in all age groups, but the prevalence of this trait waned with age. Of those in the 'fairly well' group the females ate more quickly than males at both the younger and older ages. The prevalence of this trait again tended to wane with age. Whereas in the 'somewhat' group the trait increased with age in both sexes and females ate more quickly than males at all ages.

As the younger age groups in the presence of complete traits have been shown to be more competitive

and harder driving it is not surprising that being pressed for time they had to eat more quickly.

Behavioural Pattern and IHD Prevalence

There was a significant correlation between type A behaviour and PMI and ischaemic-type ECG changes after adjustment for age and sex. However, there was a significant interaction between sex and behaviour on the prevalence of angina and ischaemic-type ECGs without symptoms. Thus separate analysis of the data of each sex was made. It revealed, in addition, a significant correlation between type A and angina in males, and ischaemic-type ECG changes without symptoms in females.

Multivariate analysis with control of other risk factors showed a significant correlation only between type A and PMI in males, and ischaemic-type ECGs in females.

These results are in line with similar results reported by several cross-sectional and prospective studies.

However, the study of the correlation of type A behaviour among women with regard to IHD has received little attention compared with studies among men. Only 5 investigators have done so (Rosenman and Friedman, 1961; Bengtsson et al., 1973; Kenigsberg et al., 1974; Shekelle et al., 1976; Haynes et al., 1978, 1980). It

is interesting to note that in a large cross-sectional study Bengtsson et al. (1973) found that women who had been hospitalized for a myocardial infarction scored significantly higher on an aggression scale than a healthy reference group. This association was not apparent among women when angina, assessed by the Rose Questionnaire (1965), was examined. However, the Framingham results (Haynes et al., 1978, 1980) suggest that there is an association with type A behaviour and angina pectoris in women.

Behaviour Pattern and Coronary Risk Factors

The age-adjusted percentage of body mass index $\geq$ 28 was higher amongst type A men than type B. The trend amongst women was reversed. In both sexes the age-adjusted percentages of hypertensives and borderline hypertensives were higher amongst type A than type B. With regard to smoking habits the percentages of current smokers, as well as ex-smokers, were higher in type A than in type B for both men and women. These findings are in agreement with previous studies suggesting that heavy cigarette smoking and the prevalence of hypertension are more common in subjects with type A behaviour (Friedman, 1979).

In both sexes the age-adjusted percentage of plasma cholesterol $\geq$ 220 mg% was higher among type A

than type B. These results concur with those of previous studies suggesting that type A behaviour may raise plasma cholesterol (Friedman et al., 1958; Thomas and Murphy, 1958; Wertlake et al., 1958; Peterson et al., 1962) and are in agreement with those studies which found that average serum cholesterol of both men (Friedman and Rosenman, 1959) and women (Rosenman and Friedman, 1961) who exhibited type A behaviour pattern was significantly higher than that of type B counterparts.

For fasting blood glucose the percentage $\geq$ 120 mg% was higher among type A in males whilst in females it was higher in type B. There are no comparable findings in the literature but type A behaviour was reported to increase insulinogenic response to glucose (Friedman et al., 1970).

The percentage of those who took exercise was higher among type A than type B in both sexes.

These findings suggest that the differences among the coronary risk factors in relation to behaviour pattern could partly account for the differences in IHD prevalence between the two types of behaviour.

However, data presented by two studies (Shekelle et al., 1976; Brand et al., 1976) showed that the type A behaviour pattern was an independent risk factor for IHD. Furthermore the data from the Framingham Study (Haynes et al., 1978, 1980) showed that type A behaviour pattern was independently associated with IHD prevalence and

incidence in both sexes when controlled for standard risk factors.

Research into the biochemical pathways that might explain the association between behaviour pattern and IHD has been carried out. In a study by Simpson et al. (1974) type A men were shown to have higher platelet counts in their whole blood as well as lengthened durations in noradrenalin-induced platelet aggregation after exercise than type B men. Type A men were distinguishable from type B men mainly by their response to the challenge of exercise. Jenkins et al. (1975) reported similar results. These findings were similar to the results of Friedman et al. (1975) who found that type As exhibit greater increases in norepinephrine concentrations than type Bs under competitive but not under resting conditions.

OCCUPATION

There were significant differences in the prevalence of IHD with age, sex and occupation. The highest IHD prevalence rates were found among the administrative staff, the intermediate rates among manual workers and the lowest among the teachers.

Relation to Coronary Risk Factors

In both sexes, the teachers had the lowest body

mass index, while the highest was among the male administrative staff and the female manual workers. Also the teachers had the lowest systolic and diastolic blood pressures in both sexes. Plasma cholesterol level was highest among male administrative staff and lowest among male manual workers, while in females it was highest among administrative staff and lowest among teachers. In both sexes, the teachers exercised the most and smoked the least.

Comparison with Grade of Employment

Indicators such as occupation, education and income are usually used to define the socio-economic status of people. The classification of the participants into three groups, which was originally done by UNRWA for organisational purposes, does not take into consideration the three indicators, except in two groups, teachers and manual workers, each consisting of a homogeneous group. However, the administrative staff are a disparate group with different educational qualifications and different employment grades. In the case of teachers, the minimum education required for their employment is a teacher training certificate or a university degree, whereas in the case of manual workers no formal education is required.

Grade of Employment and IHD Prevalence

Therefore, the comparison of IHD prevalence rates and levels of risk factors in relation to the grade of employment can only be made between teachers and manual workers.

It was noted that the prevalence of IHD among manual workers was higher than in teachers. Also teachers had lower levels of coronary risk factors than manual workers. The teachers, perhaps being aware of IHD risks, smoked less and took more exercise than the manual workers who had the least education and the lowest salaries. In females plasma cholesterol, similar to other risk factors, was lower among teachers than manual workers. In males plasma cholesterol alone of all risk factors was higher among the teachers than manual workers. These findings are in line with recent trends in the literature (Marmot et al., 1978).

It can therefore be concluded that IHD prevalence rates are inversely associated with the grade of employment and level of education.

HYPERGLYCAEMIA

The value of comparing surveys on the prevalence of hyperglycaemia and diabetes mellitus is questionable. This is due to differences in methods of measurement,

definition and diagnostic criteria used for these two conditions.

It should be pointed out that it would have been preferable in this study if measurements of insulin and post-prandial blood glucose had been made in addition to those of asting blood glucose. However, insulin estimation was not possible due to lack of facilities and the intake of a glucose load was not welcomed by the participants.

However, the measurement of fasting blood glucose served the purpose of describing its distribution in this cohort and allowed the study of the inter-relationships between IHD prevalence and other coronary risk factors and levels of fasting blood glucose. Also the baseline data will be used in the longitudinal study for the assessment of coronary risk that might be associated with asymptomatic hyperglycaemia.

The prevalence rates of hyperglycaemia rose with age in both sexes, the trend being significant for both males and females except for the oldest age group of females.

After adjustment for age and sex, the prevalence of IHD was significantly higher in those who had

hyperglycaemia in both sexes. However when multivariate analysis was used this association was no longer significant.

It should be noted that the hyperglycaemics in this study include those who reported a previous diagnosis of diabetes whether or not they were on drug therapy at entry. Also, those on anti-hypertensive medication (especially oral diuretics) were not excluded from the study cohort. It is quite probable that the inclusion of these two categories of patients in the univariate analysis has led to the observed association between IHD and hyperglycaemia. IHD is quite common among diabetics (Pell and D'Alonzo, 1970; Keen and Jarrett, 1975; West, 1978) and there is also extensive evidence for an increased prevalence of glucose intolerance among survivors of myocardial infarction (Opie and Stubbs, 1976). The observed associations between blood glucose and IHD might be caused by a decrease in glucose tolerance secondary to the preceding (subclinical) disease (Cerasi and Luft, 1977).

When the association between asymptomatic hyperglycaemia and subsequent development of IHD is examined, the diagnosed diabetics and hypertensives under treatment will be excluded from the analyses.

In the series of papers recently published by the International Collaborative Group (1979), the Olivetti Study (Farinaro et al., 1979) measured the fasting glucose only, while the others either measured post-load

blood glucose alone or preceded by fasting blood glucose. Neither in the Olivetti Study, nor in most of the other studies was a significant association between baseline asymptomatic hyperglycaemia and IHD found. Furthermore, the findings of the Chicago Heart Association Detection Project in industry (Stamler et al., 1979) indicated that when the diagnosed diabetics were included in their appropriate places in the distribution and further analysis was carried out, there was still no positive association between glucose level and subsequent coronary mortality.

The correlations between hyperglycaemia and other coronary risk factors were dealt with under the individual discussions of the latter.

ORAL CONTRACEPTIVES

The extent of the use of oral contraceptives varies widely from country to country. In Jordan oral contraceptives became available in the mid-1960s. They are becoming increasingly popular among women of reproductive age.

The Ministry of Health in Jordan has introduced family planning services at maternal and child health centres in the cities. They offer a full range of contraception. Furthermore, oral contraceptives are available without prescription in all chemists' shops

in the country and are commercially advertised. Although there is evidence that there has been an increase in sales of oral contraceptives no reliable data were available.

IHD Prevalence and Oral Contraceptives

There were no significant differences in the prevalence of angina or possible myocardial infarction with age and usage of oral contraceptives. However, there were significant differences in the prevalence of ischaemic-type ECG changes ($P < 0.01$) and ECG changes without symptoms ($P < 0.05$) with age and usage of oral contraceptives.

On the other hand, when the oral contraceptive users and ex-users were combined into a single category, in order to overcome the small number problem, the age-adjusted prevalence rates for all forms of IHD in this study were found to be significantly higher among users and ex-users than those who had never used oral contraceptives.

Limitations of Data

In interpreting the difference in the prevalence of IHD in those using oral contraceptives (users), those who stopped using them (ex-users) and those who had never

used them, the following should be taken into consideration. Firstly, since small numbers of users, ex-users and IHD cases were involved, firm conclusions cannot be made. Without more data it was not possible to examine the inter-relationships between age, smoking, duration of oral contraceptive use and risk of IHD, nor to compare the effect of continuous oral contraceptive use with intermittent use. Secondly, the relationship of IHD prevalence to the type and dose of oestrogen and progestagen chould not be examined, because of the large number of preparations available in the local market.

Inman et al. (1970) showed the influence of the oestrogen content in the pill on the frequency of thromboembolism. It has been shown that the relative risk of developing thromboembolic disease is reduced with the use of pills containing low doses of oestrogen (Stolley et al., 1975), but it is not clear that all vascular complications of the pill are related to oestrogen dose (Fregly, 1974; Weir et al., 1974; Mann et al., 1975).

Also the oestrogenic potency of the pill depends not only on the dose and type of oestrogen, but also on the dose and type of progestagen, thus a reduction in oestrogen dose does not invariably lead to an equivalent reduction in oestrogenic potency (Chihal et al., 1975). The Royal College of General Practitioners Study (1977) related the incidence of hypertension to the dose of progestagen in the pill.

Thirdly, whether the observed relationship between the pill and IHD is causal or attributable to some other factor or factors which are correlated with pill use is unclear. It may be argued that the recent changes in female employment, education, smoking habits and life style may have contributed to the observed IHD prevalence among young women. The effect of employment on the development of vascular diseases in females has not been studied in detail. However there is evidence that working women suffer excessive cardiovascular morbidity and mortality (Moriyama et al., 1971). The effect of changes in life style are very difficult to measure.

In the Oxford Study (Vessey et al., 1976) and the Royal College of General Practitioners Study (1977) oral contraceptive use was associated with cigarette smoking.

Although raised plasma cholesterol levels have been shown to be associated with IHD, patients who are known to have hypercholesterolaemia are likely to be advised to use some other form of contraception.

With so many factors associated with IHD it may be necessary to examine the independent risk of each factor and to consider whether any of them are secondary, arising because the risk factors are themselves interrelated. However, as the data are cross-sectional and due to the small number of pill users and ex-users the calculation of the independent contribution of oral contraceptives was not made.

Fourth, it is also possible that the observed correlations were part of some non-specific phenomenon, and that similar relationships exist for other apparently unrelated diseases. This also was not examined.

Fifthly, the cultural aspect should not be overlooked among Jordanian women. There are social barriers that restrict discussions relating to their personal practices. Thus there was probably an under-reporting of the usage of oral contraceptives among women in this study. Consequently no further analyses were done and the data reported should be interpreted with caution.

ALCOHOL

The results of this study show that there is a significant association between alcohol intake and IHD. However, in view of the small numbers involved and limitations of the data collected no firm conclusions can be drawn and hence no further analysis has been undertaken.

Findings of other studies differ on the relationship between alcohol consumption and IHD but the majority show that the former may protect against the latter.

However, alcohol consumption may indirectly be associated with IHD in physically inactive people. It may also encourage cigarette smoking, and by increasing

the calorific input it may contribute to excess weight.

In Jordan, alcohol and spirits are sold in drink shops and supermarkets owned by Christians. They are also served in licensed public establishments, such as bars, restaurants and hotels. They are also imported from several other countries, besides being produced in Jordanian distilleries.

In accordance with their religion, Moslems must not sell, serve or drink alcohol or spirits. This, however, is less than strictly observed by some Moslems in Jordan.

Although formal data on alcohol consumption are not available the indications are that the problem of alcoholism in Jordan is not to be underestimated. Edwards (1979) however has recently reviewed the spread of alcoholism in developing countries and has concluded that "alcoholism has a serious impact in these countries".

Rapid urbanization usually brings about social change and this can lead to a weakening of cultural restrictions on alcohol drinking.

The participants in the study included Moslems and Christians but the bulk consisted of Moslems. Thus it is probable that the data collected on alcohol consumption, particularly from women, were influenced by the local religious codes and habits and were therefore conservative; consequently the results should be interpreted with caution.

RISK FACTORS AND IHD PREVALENCE

In this study, as in several other studies, the risk factors which are shown to have significant correlation with IHD prevalence are hypercholesterolaemia, hypertension, smoking, type A behaviour and physical inactivity. Diet and hyperglycaemia failed to show consistent relationship to IHD. There was, however, a significant inverse correlation between brown bread intake and IHD prevalence. The important question which will be discussed here is, does the prevalence of risk factors account for the observed IHD prevalence?

A study of the prevalence of IHD risk factors has not previously been done in Jordan. Also reliable data for per capita consumption of specific nutrients, such as saturated fats and eggs, are not available. Therefore, it was not possible to discuss the observed prevalence of IHD in relation to these parameters.

An alternative means of relating risk factors to IHD may be investigated through mathematical models of which the most widely applied is the multiple logistic function. No such model is available for any of the Arab populations. Even if such a data base was available, it could hardly be generalized to other populations. Another problem lies in the fact that we are dealing with cross-sectional data. Thus, no comparison can be made between predicted and observed IHD prevalence.

In a cross-sectional study the characteristics observed at one point in time may be different from those

which existed during the formative period of the underlying disease process. The effect of a risk factor such as cigarette smoking may depend on the dose and period of exposure. Thus, the periods covered by IHD prevalence and risk factor changes may not be identical, nor can the possible lags between changes in risk factors and their effect on IHD be accurately taken into consideration.

With cigarette smoking, available evidence indicates that although IHD mortality begins to decline within a few years of cessation, the full benefit may not be realized until 5 or 10 years after giving it up (Hammond, 1966). With cholesterol lowering, little is known about how rapidly the benefits may be realized.

On the other hand, there may be an under-estimation of the observed prevalence of some of the risk factors. As the study population consisted of staff who underwent regular in-service medical examination and had access to free medical service, it was very likely that a percentage of the hypertensive and hypercholesterolaemic subjects were diagnosed, treated and controlled.

Similarly, a potential problem with data based on smoking behaviour is the systematic under-reporting by survey participants. In the U.S.A., for example, there is evidence that such under-reporting may have increased in recent years (Warner, 1977). To overcome this problem, trends in per capita cigarette consumption

(defined as total domestic cigarette sales divided by the population of the country above 17 years of age) are examined. Neither are data on cigarette consumption available, nor have surveys on the cigarette smoking habits of the Jordanian public been done.

Atherosclerosis and IHD, in particular, appear to have a multifactorial origin. Thus, at any level of 'risk factors' for example, total cholesterol, risk varies over a wide range depending on the dose of other associated risk factors. Even within the range generally conceded to be 'normal' for these factors, risk varies over a fairly wide range (Kannel, 1974).

Some groups known to be at very high risk may in fact show fewer cases of disease than a much larger group with only slight to moderate increase in risk. For example, with familial hypercholesterolaemia the individual attributable risk of premature death is high, but because the prevalence of this condition is very low, such persons amount to less than 1% of all IHD cases or in other words the population attributable risk is very low.

On the other hand at moderate elevation of serum cholesterol levels, the individual risk is much lower but the population prevalence is far higher. In line with this discussion, the bulk of IHD in the Framingham Study (Kannel et al., 1979) has been shown to arise out of the segment of the population with only

modest elevations of cholesterol. Thus it is not surprising that the serum total cholesterol levels in this survey showed relatively modest elevations in relation to IHD prevalence.

Investigations on a cross-sectional population base have been made in several studies to see to what extent the relationship between diet and serum cholesterol carries over to the total populations. Both the Framingham Study (Kannel and Gordon, 1970) and the Tecumseh Study (Nichols et al., 1976) used careful dietary data collection methods. Correlations were examined between dietary lipid intake and serum cholesterol measured at the time the dietary history was taken. Zero or near zero correlations were found between the various components of diet and serum cholesterol level. Jacobs et al. (1979) examined the consistency of the findings of these cross-sectional studies of zero correlations with the results of controlled dietary experiments. They concluded that the confounding that resulted from the uncontrolled conditions under which most epidemiological observations were made, was sufficient to undermine their validity with respect to investigation of the relationship between diet and serum cholesterol. It was also concluded that cross-sectional designs were not suitable for studying this relationship.

A lack of accosiation between certain risk factors and IHD prevalence has been shown in many cross-sectional studies, for example:

(a) In the Tecumseh Study (Epstein et al., 1965), no association was found between smoking and IHD prevalence. There was also lack of statistical significance for the relation between serum cholesterol level and IHD prevalence.

(b) In the Busselton Study (Welborn et al., 1969), no significant association was found with overweight nor with current cigarette smoking and IHD prevalence.

(c) In the Chandigarh Study (Sarvotham and Berry, 1968), no correlation was found between smoking and prevalence of IHD.

(d) In prevalence comparisons in Europe (Rose et al., 1968), there was no evident correlation between smoking and rates for the various manifestations of IHD. The mean blood pressure levels for individual surveys do not seem to correlate with indices of IHD.

(e) In the Jamaican studies (Florey et al., 1972, 1973; Florey, 1978), no relationship was found between blood sugar level and signs and symptoms of cardiac ischaemia (based on answers to standard chest pain questionnaire and ECG abnormalities, Minnesota codes I 1-3, IV 1-3, VI 3, VII 1), nor between blood sugar or diastolic pressure in women. Although the mean values and distributions of serum cholesterol, blood pressure, and blood sugar in Jamaica were

similar to those found in studies of populations living in developed countries (Miall and Cochrane, 1961; Florey et al., 1973); in this developing population clinical IHD was rare and the inter-relationships between the risk factors seen in other studies were either less strong or could not be detected.

The considerations presented here seem to indicate that in a cross-sectional study the observed prevalence of traditional risk factors appears to explain only a part of the IHD prevalence and probably does not account for all of it. The confounding factors and unmeasured variables may obscure relationships between the risk factors and IHD.

There were probably other unknown and unmeasured factors, because there were persons who did not have any of the well-established risk factors and who developed IHD at an early age.

The other unmeasured factors which one may consider and which have not been investigated by the survey are first, the cross-cultural problems of the study cohort, and second, the measurement of cholesterol-bearing lipoproteins. During the last three decades as a result of their work with UNRWA and rise in the standard of living in Jordan the participants underwent improvement in their standard of living. Thus, this cross-cultural change during adulthood may have left its

markson the cardiovascular health of this cohort. It is reasonable to assume that men and women between 30 and 60 years old were the group most involved in "acculturated" life style and accompanying conflicts and thus had a high IHD prevalence.

Also plasma lipoproteins were not measured and therefore the association between the lipoprotein components and IHD prevalence was not studied. Data from the Framingham study and elsewhere (Kannel et al., 1979) have shown that the risk of IHD in persons younger than 50 is strikingly related to the serum total cholesterol level. The contribution of the serum total cholesterol to risk has been found to be determined by its partition into various lipoprotein fractions. A relatively large amount of cholesterol in the low-density lipoprotein fraction is atherogenic, whereas that in the high-density fraction appears to be protective.

The independent contribution of very-low-density lipoprotein and its triglyceride or cholesterol content has, on the other hand, not been established. Kannel et al. (1979) concluded that the previous assumption, that almost all of the lipid information "pertaining to CHD resided in the serum total cholesterol", must be accordingly modified.

The lipoproteins merit careful consideration both in identifying coronary candidates and in designing preventive measures to reduce risk. Therefore it would

have been relevant to study the association of these fractions with IHD prevalence, but due to lack of facilities this was not possible.

However, if the IHD determinants do not seem to account for the entire prevalence observed, then we must turn our attention to the impact of secondary prevention and the case fatality as possible explanations for the high prevalence.

Although it is likely that both the case fatality of IHD in Jordan is low and the disease occurs in very mild forms with generally built-in benign prognosis, the data to confirm this were not available.

It may be argued that the advent of pre-hospital emergency medical services, coronary care units and coronary artery bypass surgery in Jordan has prolonged the lives of patients with IHD and therefore lowered the case fatality rate.

There is considerable evidence that in developed countries medical and surgical treatment had small impact on the burden of IHD in the community (Joint Working Party, 1975). In Jordan it is unlikely that there will be a different outcome. Nevertheless, in order to determine whether or not these services do prolong the lives of IHD cases, it is necessary to know what proportion of the observed IHD cases were covered by such programmes and whether or not these programmes have had a significant impact on IHD mortality.

There is not much evidence to support the argument for the high IHD prevalence noted in this study being due to low case fatality or effective secondary prevention.

In conclusion, it is not possible in a cross-sectional study to quantify the relative extent to which the high IHD prevalence has been due to risk factors and the extent to which it has been due to other causes.

CHAPTER VII

CONCLUSIONS

Putting IHD on the Map of Developing Countries

The epidemic of IHD was shown to be spreading to a comparatively wealthy cohort of a developing country. IHD is usually considered to be prevalent in developed countries and rare in developing countries, but it appears that the change in the distribution of the disease has begun. This is particularly so where people in developing countries adopt life styles similar to those in developed countries.

In the years which followed the Second World War, IHD emerged as the major disease in the developed countries. In more recent years the mortality from IHD in U.S.A. and Australia has declined (Walker, 1977; Christie, 1974). In the U.K. the rate has become fairly stable in the general population (Marmot et al., 1978), however there has been shown to be a decline among doctors (Doll and Peto, 1976).

It appears that there has been a change in the frequency and distribution of this disease among social classes. In England and Wales the rise in IHD this century first affected people in the upper social classes and the disease has latterly become progressively more common in the lower social classes (Marmot et al., 1978).

It might reasonably be assumed that IHD would have been a problem first for the affluent and educated groups in the society such as doctors, lawyers and executives, and only in recent years for the non-affluent unskilled workers who as a result of mechanisation and decrease in income gradient along social class scale have lost the protective effect of strenuous occupation and poor diet. They also smoked more and took less leisure exercise than those in the upper social class. The impact of modern life may also have contributed to the increased vulnerability of low social class people.

On the basis of the change in the distribution of IHD in developed countries, can we expect a similar change in distribution between developed and developing countries?

Hard data on the frequency and distribution of IHD in developing countries are sketchy. But there are indications that the prevalence of this disease is increasing in these countries (Sarvotham and Berry, 1968; Mial et al., 1972; Tangchai, 1972; Badran and Sorour, 1972; Massoud et al., 1978; Hassan and Wasfi, 1972; Emara and Al-Yousuf, 1977). This has prompted WHO to warn that the IHD threatens to become "mankind's greatest epidemic" (WHO, 1969).

IHD and Behaviour Patterns

The results of this survey underline the importance

of type A behaviour pattern as a risk factor to IHD and physical exercise and fibre intake (brown bread) as protective factors against IHD. It also confirms the role of elevated cholesterol, hypertension and cigarette smoking in the development of IHD.

Current opinion is that IHD is caused by multiple risk factors that are probably additive. However as most of these factors are related to behaviour and due to noxious habit patterns, it seems prudent to hypothesise that IHD is caused by incongruent behaviour.

The crucial question which arises is, can behaviour be changed? and if the answer is yes, would such change produce protective effects? For example, would modification of diet lower serum cholesterol? Could we encourage those whose needs are great for surveillance, such as hypertensives, to make full use of the services and treatment available? Is it possible to change the norms of society so that cigarette smoking becomes a bad habit?

There are no definite answers to these questions but there is encouraging evidence.

The recent decline in IHD mortality in the U.S.A. has coincided with changes in life style of the population. Recommendations for preventive action against IHD and its risk factors were followed by modification in diet, reduction in cigarette consumption and increased exercise (Stamler, 1979).

The Finnish Mental Hospitals Study showed that a

reduction in serum cholesterol by fat-modified diet reduced IHD mortality (Miettinen et al., 1972). The Los Angeles Veterans Administration Domiciliary Facility Study (Dayton et al., 1969) showed similar results.

As to ceasing cigarette smoking, the 22 centres of the Multiple Risk Factor Intervention Trial (MRFIT Research Group, 1977, 1978) have shown that it is possible to effect and sustain stopping of cigarette smoking in a sizeable proportion of middle-aged coronary-prone men. With regard to the effect of stopping smoking on the subsequent development of IHD, while no data are available from a randomized controlled trial, data from prospective studies showed that it reduces the risk of IHD incidence and mortality (Hammond and Garfinkel, 1969; Pooling Project Research Group, 1978).

With regard to lowering elevated blood pressure the Veterans Administration trial of antihypertensive drugs (1967, 1970, 1972) and the Hyptertension Detection and Follow-up Programme (1977, 1978) showed that it is possible to control this risk factor. The Veterans Administration trial also showed a positive, though statistically not significant, trend towards reduction of IHD.

With regard to modification of type A behaviour, Friedman (1979) suggests that it could be modified. No data from randomized controlled trials are available on this matter.

The behavioural changes required for risk reduction have been shown to be potentially achievable at least by individuals. People do stop smoking, maintain normal blood pressure, cut down on their intake of saturated fats, dietary cholesterol, sugar and salt, they do reduce their weight to an optimal level and take habitual exercise. Some even achieve success in bringing their daily tensions under control (Inter-society Commission for Heart Disease Resources, 1970, 1972; Blackburn, 1974; Olefsky, 1974; Stamler, 1979).

The problem remains as to how best to convey behavioural changes programmes to heterogeneous populations in a successful and cost-effective way. The community-model rather than the health-centre model would provide the suitable milieu in which social support would become part of the programme. As smoking has been prohibited in many social and transport facilities those who have been persuaded to change this habit could gain social reinforcement. Also the provision of more facilities for leisure time activity should encourage people to take more exercise. If protective diets recommended by scientific bodies were subsidised by governments, this could favourably affect people's dietary habits.

The Need for Research

It appears that the prevalence of IHD observed cannot be fully explained by the prevalence of established risk factors. It is probable that there are some other risk factors which act adversely in this cohort. Therefore there is a need for more research into the coronary risk factors to identify the missing links between IHD and its antecedents. Hard data are required to explain why much of the international differences in IHD incidence and prevalence are not accounted for by differences in the established risk factors.

There is also need for research into the physiological and biological mechanisms of coronary risk factors in order to explain more adequately the associations between these factors and IHD.

As it is beyond the scope of this thesis to elaborate on the areas of research required in each of the risk factors, only the areas of research into one of the risk factors underlined by this study, i.e. physical activity, will be outlined briefly. There are four areas where more research is required in the field of physical activity. First, to develop an assessment procedure for physical activity which takes into consideration the heterogeneity of the forms of exercise and variations between intensity of exercise when performed at different ages. Second, the kind and amount of exercise that is desirable to prevent IHD. Third, what kinds of physical

activity are suitable for the coronary patients? Fourth, if vigorous exercise is protective against IHD, what mechanisms are involved?

The Need for Standardization of Methodology

Variation in population sampling, survey design, definitions, diagnostic criteria, laboratory methods, instrumental measurements and research techniques limits the value of inter-survey comparisons. These differences have been noted both between and within individual countries. This makes it more difficult to pool results from different surveys or to make comparisons between studies.

The questionnaires commonly used for the study of IHD incidence and prevalence have been developed mainly for use in Western countries. Their translation and application in different cultures may have some effect on their validity.

Therefore, there is need for standardization of methods, training based on standard material, and field conditions; also quality control of laboratory techniques and measurements.

The Need for Prevention

The level of prevalence observed in this study identifies a great need for action. Emphasis should be

placed on prevention rather than cure.

There is no cure for the disease. Also we cannot by medical or surgical intervention or secondary prevention, reverse the underlying disease process in patients already known to have the disease.

Despite the remarkable progress which has been made in understanding the processes involved in IHD, a coronary heart attack can kill a person before he can reach or is reached by medical care.

The studies of Oxford, Edinburgh and London of coronary heart attacks have shown that the opportunities of the medical intervention to save or spare lives of coronary victims are very limited (Kinlen, 1969; Armstrong et al., 1972; Pedoe, 1975). In these studies about two-thirds of fatal attacks occurred at home or at work and were medically unattended. Only about one-third occurred in hospital.

Coronary Care Units with resuscitative equipment have been established in large teaching hospitals, artificial pacemakers are available for control of heartbeat, rhythm abnormalities can be monitored and controlled, diseased coronary arteries can be bypassed. Despite all these formidable and costly services the process of atherosclerosis and the damage to the heart muscle are irreversible.

The results of secondary prevention trials to lower plasma lipids by clofibrate and nicotinic acid have

been disappointing. Neither drug produced significant reduction in IHD mortality and both drugs were associated with complications and increased incidence of cardiovascular events (Coronary Drug Project, 1975).

A number of primary prevention trials have also been carried out. The hypothesis in these studies is that reduction of the risk factors will reduce the incidence of and mortality from IHD. Some of these trials have been conducted among institutionalized groups; these include the Los Angeles Administration Domiciliary Facility Study (Dayton et al., 1969). the National Diet-Heart Study in Fairbault, Minnesota (1968), and the Finnish Mental Hospital Study (Miettinen et al., 1972). Others have been conducted in large groups of free-living people; these include the New York Anti-Coronary Club (Rinzler, 1968), the Chicago Coronary Prevention Evaluation Programme (Stamler et al., 1976; Stamler, 1977), Studies of persons with diabetes (Stone and Conner, 1963; Kiehn et al., 1978) and the Oslo primary and secondary prevention diet trials (Leren, 1966; Hjermann, 1977). Despite the limitations of these studies they have provided some evidence that multifactorial intervention may produce reduction in IHD incidence and mortality.

Two important multifactorial trials are now underway in the U.S.A. and Europe to establish the effectiveness of multifactorial primary prevention through modification of risk factors. In the MRFIT trial

selected subjects at high risk of heart attack are randomized into special and usual-care groups. In the WHO European Collaborative Group (1974) communities or groups rather than individuals are randomized into treatment or control groups. The final results of these studies are expected to clarify the efficacy of reducing coronary risk factors and the effect on IHD incidence and mortality.

As the effect of medical and surgical treatment in reducing IHD burden is small and as the cost of inaction is formidable, this has prompted a number of scientific bodies to recommend measures aimed at preventing the disease. The most notable of these is the Joint Working Party (1975) which emphasized the multifactorial concept of coronary risk and made very sound recommendations.

In conclusion it is evident that primary prevention must be our ultimate goal. Given the limitations in resources, this is clearly the most cost-effective approach.

Postscript

It should be stressed that the results presented in this thesis are based on cross-sectional data and all the reservations that pertain to inferences from prevalence data must be borne in mind.

the UNRWA Study is a selected cohort and extrapolation to the general population must be done with caution.

TABLE 1

Prevalence of Coronary Heart Disease (CHD) by Age and Sex; Tecumseh (Epstein et.al., 1965)

Age	Total	"Probable" CHD						"Suspect" CHD	
			Prev-alence		Prev-alence		Prev-alence		Prev-alence
yr	no.	no.	no./1000 pop.	no.	no./1000 pop.	no.	no./1000 pop.	no.	no./1000 pop.
Men									
16-29	667	2	3	1	1	3	4	28	42
30-39	679	2	3	5	7	7	10	45	66
40-49	467	10	21	6	13	16	34	77	165
50-59	331	29	88	11	33	40	121	64	193
60-69	178	18	101	14	79	32	180	43	242
70+	127	12	94	11	87	23	181	46	362
Total	2449	73	30	48	20	121	49	303	124
Women									
16-29	819	1	1	0	0	1	1	64	78
30-39	702	2	3	1	1	3	4	81	115
40-49	474	5	11	3	6	8	17	80	169
50-59	327	10	31	10	31	20	61	86	263
60-69	196	21	107	11	56	32	163	53	270
70+	162	12	74	12	74	24	148	53	327
Total	2680	51	19	37	14	88	33	417	156

TABLE 2

CHD Prevalence by Age-group, Sex and Race in Evans County (McDonough et.al., 1965)

	Age Group	Definite	Probable	Possible	Negative	Total	Ratio*
White males	40-49	6	-	6	242	254	24
	50-59	10	3	6	200	219	59
	60-69	14	5	10	110	139	137
	70-74	3	3	5	41	52	115
	Total	33	11	27	593	664	68
Negro males	40-49	1	-	1	148	150	7
	50-59	-	2	3	128	133	15
	60-69	2	2	3	78	85	47
	70-74	-	1	4	22	27	37
	Total	3	5	11	376	395	21
White females	40-49	-	-	3	236	239	-
	50-59	1	1	7	198	207	10
	60-69	2	3	5	169	179	28
	70-74	-	2	1	57	60	33
	Total	3	6	16	660	685	12
Negro females	40-49	-	1	2	178	181	6
	50-59	2	1	3	139	145	21
	60-69	-	-	5	96	101	-
	70-74	1	-	-	44	45	22
	Total	3	2	10	457	472	11
Total	40-49	7	1	12	804	824	10
	50-59	13	7	19	665	704	28
	60-69	18	10	23	453	504	56
	70-74	4	6	10	164	184	54
	Total	42	24	64	2086	2216	30

*Definite plus probable cases per 1000 examined. Ratio for the age-group 40-74 are age-adjusted (indirect method).

TABLE 3

Prevalence of various symptom categories and their relationship to ECG findings by age, Whitehall Study (Rose et.al. 1977).

Symptom category		40-49 years	50-59 years	60-64 years	All ages adjusted
Study group	total	7732	8384	2387	18403
	ECG +	278 (3.6)	609 (7.4)	264 (11.1)	1151 (6.3)
Angina grade 1	total	204 (2.6)	365 (4.4)	155 (6.5)	719 (3.9)
	also ECG +	18 (9.0)	59 (16.0)	31 (20.0)	108 (14.0)
grade 2	total	27 (0.4)	93 (1.1)	44 (1.9)	164 (0.9)
	also ECG +	3 (11.0)	21 (23.0)	15 (34.0)	39 (19.0)
Possible infarction	total	422 (5.5)	612 (7.4)	197 (8.3)	1231 (6.7)
	also ECG +	16 (4.0)	104 (17.0)	43 (22.0)	163 (12.0)
Intermittent claudication	total	47 (0.6)	68 (0.8)	32 (1.4)	147 (0.8)
	also ECG +	0 (0.0)	4 (6.0)	5 (16.0)	9 (5.0)

ECG+ is Minnesota Code 1.1-3 or 4.1-4 or 5.1-3 or 7.1.
Per-entages are given in parentheses.

TABLE 4

Prevalence of coronary heart disease (CHD) by age and sex in Busselton, (Welborn et.al., 1969).

Age Years	"Probable" CHD									"Suspect" CHD	
	Total	Class 1 Prevalence		Class 2 Prevalence		Class 3 Prevalence		Total Prevalence		No	No/10
	No	No	No/1000	No	No/1000	No	No/1000	No	No/1000		
Men											
21-29	257	0	0	1	4	0	0	1	4	11	43
30-39	281	2	7	1	4	1	4	4	14	14	50
40-49	348	6	17	6	17	3	9	15	43	25	72
50-59	313	21	67	4	13	0	0	25	80	18	58
60-69	265	26	98	7	26	5	19	38	144	21	79
70+	174	16	92	12	69	0	0	28	161	16	92
Total	1638	71	44	31	19	9	5	111	68	105	64
Women											
21-29	273	0	0	0	0	1	4	1	4	11	40
30-39	322	1	3	5	15	0	0	6	18	12	37
40-49	355	2	6	3	8	0	0	5	14	25	70
50-59	322	10	31	7	22	3	9	20	62	21	65
60-60	248	18	73	8	32	4	16	30	121	23	93
70+	173	19	110	12	69	5	30	36	210	25	144
Total	1693	50	30	35	21	13	8	98	58	117	69

TABLE 5

The Prevalence of Coronary Heart Disease by Age and Sex in the Population Studied in Chandigarh, Sarvotham and Berry, 1968. (Corrected Rate for Women in Parentheses)

Age (yr)	Total No.	Probable coronary heart disease						Suspect CHD	
		Class I		Class II		Total			
		No.	Prevalence (no./1000 pop)	No.	Prevalence (no./1000 pop)	No.	Prevalence (no./1000 pop)	No.	Prevalence (no/1000 pop
Men									
30.1 - 40	658	7	10.6	9	13.7	16	24.3	16	24.3
40.1 - 50	344	17	49.5	5	14.5	22	64.0	9	26.2
50.1 - 60	231	16	69.3	14	60.6	30	129.9	15	64.9
60.1 - 70	96	10	105.2	2	20.8	12	126.0	11	115.6
70.1 and above	32	7	218.9	2	62.5	9	281.4	2	62.5
Total	1361	57	41.9	32	23.5	89	65.4	53	38.8
Women									
30.1 - 40	298	2	6.7	17	57.1	19 (-10)	63.7 (30.2)	32	107.4
40.1 - 50	185	3	16.2	6	32.4	9 (- 3)	48.6 (32.4)	16	86.5
50.1 - 60	117	1	8.6	6	51.3	7	59.8	20	170.9
60.1 - 70	41	3	73.2	2	48.8	5	122.0	5	122.0
70.1 and above	28	4	142.9	1	35.7	5	179.6	4	142.9
Total	669	13	19.4	32	47.8	45 (-13)	67.2 (47.8)	77	115.1

TABLE 5

The Prevalence of Coronary Heart Disease by Age and Sex in the Population Studied in Chandigarh, Sarvotham and Berry, 1968. (Corrected Rate for Women in Parentheses)

Age (yr)	Total No.	Probable coronary heart disease						Suspect CHD	
		Class I		Class II		Total			
		No.	Prevalence (no./1000 pop)	No.	Prevalence (no./1000 pop)	No.	Prevalence (no./1000 pop)	No.	Prevalence (no/1000 pop
Men									
30.1 - 40	658	7	10.6	9	13.7	16	24.3	16	24.3
40.1 - 50	344	17	49.5	5	14.5	22	64.0	9	26.2
50.1 - 60	231	16	69.3	14	60.6	30	129.9	15	64.9
60.1 - 70	96	10	105.2	2	20.8	12	126.0	11	115.6
70.1 and above	32	7	218.9	2	62.5	9	281.4	2	62.5
Total	1361	57	41.9	32	23.5	89	65.4	53	38.8
Women									
30.1 - 40	298	2	6.7	17	57.1	19 (-10)	63.7 (30.2)	32	107.4
40.1 - 50	185	3	16.2	6	32.4	9 (- 3)	48.6 (32.4)	16	86.5
50.1 - 60	117	1	8.6	6	51.3	7	59.8	20	170.9
60.1 - 70	41	3	73.2	2	48.8	5	122.0	5	122.0
70.1 and above	28	4	142.9	1	35.7	5	179.6	4	142.9
Total	669	13	19.4	32	47.8	45 (-13)	67.2 (47.8)	77	115.1

TABLE 6

PREVALENCE OF IHD IN SUBJECTS 35-64 YEARS,
LAWRENCE TAVERN, JAMAICA (Mial et al, 1972)

		MALES			FEMALES	
Symptoms	No.	E.C.G.-myocardial ischaemia*		No.	E.C.G.-myocardial ischaemia	
Possible myocardial infarction	10	4		10	2	
Possible angina	40	12	33%	51	18	34%
Both	5	2		4	2	
Neither	472	43	9%	476	71	15%

*Minnesota Code Items 1:1-3, 4:1-3, 5:1-3 or 7:1

N.B. Males 8.5% had angina and 2.9% had PMI.
Females 10.2% had angina and 2.6% had PMI.

TABLE 7

Prevalence of caridiovascular disease at entry (Rate = the crude rate per 10,000 men at risk; infarct code 04 = old infarction by history but no ECG confirmation; CHD code 09 = probable CHD, all men 40-59 at entry). (Keys, 1980)

Item	U.S. RR	Finland	Croatia	Italy	Greece	Zutphen	Serbia	Japan	Total
Men at risk	2,571	1,677	1,367	2,480	1,215	878	1,565	1,010	12,763
Definite old infarction									
Cases	61	20	4	18	4	9	9	5	130
Rate	237	119	29	73	32	103	58	50	102
Infarct code 0+									
Cases	29	15	0	4	1	2	2	0	53
Rate	113	89	0	16	8	23	13	0	41
Angina pectoris									
Cases	25	22	1	5	2	3	5	0	63
Rate	97	131	7	20	16	34	32	0	49
CHD code 09									
Cases	2	0	1	0	0	0	4	0	7
Rate	8	0	7	0	0	0	26	0	5
Other heart disease									
Cases	58	44	16	44	17	16	28	4	227
Rate	226	262	117	177	140	182	179	40	178
Hypertensive vascular disease									
Cases	67	27	2	1	12	0	18	0	127
Rate	261	161	15	4	99	0	116	0	100
Peripheral vascular disease									
Cases	9	13	0	4	0	3	15	0	44
Rate	35	78	0	16	0	34	96	0	34
Cerebrovascular disease									
Cases	5	4	1	3	0	0	1	0	14
Rate	19	24	7	12	0	0	6	0	11

TABLE 8

AGE-ADJUSTED PREVALENCE RATES FOR 4 DEFINED SYNDROMES (ROSE et.al., 1968)

Survey	PREVALENCE (%)			
	Chest pain or discomfort	Angina of effort	History of possible infarction	Intermittent Claudication
Brussels	33	4.9	2.4	3.2
The Hague	29	6.3	1.5	2.2
Milan	40	2.7	3.0	2.2
Moscow	63	4.4	9.6	2.1
Naples	24	3.0	3.6	0.5
Odense	35	0.3	3.9	0.4

TABLE 9

Prevalence of coronary heart disease as determined by ECG and standard questionnaire for Japanese males by geographic location (Marmot et.al., 1975)

Observational base and diagnosis	Age-adjusted prevalence/1000		
	Japan	Hawaii	California
ECG			
Definite CHD*	5.3	5.2	10.8
Definite and possible CHD+	25.4	34.7	44.6
Questionnaire			
Angina pectoris‡	11.2	14.3	25.3
Possible infaction‡	7.3	13.2	31.4
(No. of men)	(2141)	(8003)	(1834)

* Major Q/QS abormalities: Minnesota Codes 1-1-1 through 1-1-7.

\+ Definite and possible CHD = major and minor Q/QS abnormalities: Minnesota Codes 1-1-1 through 1-3-6.

‡ Cardiovascular questionnaire (Rose and Blackburn, 1968).

The numbers of men in each of the tables vary due to differences in the numbers of missing values.

TABLE 10

Standardized Mortality Ratios for CHD among California Seventh Day Adventist Males age 35 and over by current dietary habits, 1960 - 1965

	Pure vegetarian	Lactoovo-vegetarian	Non-vegetarian
Standardized mortality ratio	14	39	56
Observed deaths	2	120	141

Source Phillips et al, CHD mortality among Seventh Day Adventists with differing dietary habits, Amer. J. Clin. Nutr. 31, S191-S198 (1978).

TABLE 11

TRIALS OF CHD PREVENTION BY DIET

Author	Type trial	Number of men	Mean age years	Duration, years	Serum Cholesterol, per cent change	Result
Rose et.al.(1965)	2^{ry}	80	55	2	(-0.5 mmol per l)	Negative
Research Committee(1965)	2^{ry}	264	65	3	- 6.6	Negative
Leren (1966)	1^{ry} & 2^{ry}	412	56	5	- 14	Less CHD in under 60's
Research Committee (1968)	2^{ry}	393	60	4	- 12	Negative
Dayton et.al.(1972)	1^{ry} & 2^{ry}	846	65.5	8	- 13	Less CHD and C-VD* in under 65's
Miettinen et.al.(1972)	1^{ry} & 2^{ry}	4178	50	6	- 16	CHD mortality halved.

*Cerebro-vasulcar disease

Conversion: traditional to SI units - Cholesterol 1mg/100 ml = 0.026/l

TABLE 12

Mean Cholesterol Levels in Children by Age[a]

Investigators	Sex	Age	Cholesterol (mg%)
Harrison & Peat (1975)	M/F	Cord blood	98.5 ± 22.1
	M/F	5th day	146.6 ± 24.6
	M/F	8th day	154.8 ± 24.3
Beal (1970)	M/F	1 - 6 years	168 (95 - 246 range)
	M/F	6 - 14 years	167 (107 - 249 range)
	M	14 - 18 years	156 (155 - 206 range)
Wiese et al (1966)	M/F	1 - 5 years	156 ± 38
	M/F	5 - 10 years	157 ± 27
	M/F	10 - 15 years	172 ± 22
Owen et al (1971)	M/F	1 - 2 years	159
	M/F	2 - 6 years	165
Clarke et al (1970)	M/F	16 - 18 years	166 ± 30
Baker et al (1967)	M/F	10 - 13 years	154 ± 32.5
Schilling et al (1964)	M	16 - 20 years	199 ± 3.6
Friedman & Goldberg (1975)	M/F	2 - 4 months breast fed	146 ± 4.6
	M/F	12 months breast fed	145.8 ± 1.5
	M/F	18 - 24 months breast fed	155.8 ± 6.0
	M/F	2 - 4 months formula fed	123.8 ± 8.3
	M/F	12 months formula fed	141.4 ± 1.6
	M/F	18 - 24 months formula fed	154.6 ± 4.5
	M	15 - 19 years	156.2 ± 7.9
	F	15 - 19 years	154.9 ± 5.7
Glueck & Tsang (1972)	M/F	12 months	129
Rosanen (1977)	M	5 - 19 years	234
Leonard et al (1976)	M	2 - 13 years	166.3 ± 38.6
Hennekens et al (1976)	M/F	1 - 21 years (children of healthy men)	176.6 ± 27.9
	M/F	1 - 21 years (children of men affected by MI)	185.1 ± 45
Hodges & Krehl (1965)	M	14 - 19 years	160 ± 34
Frerich et al (1977)	M/F	5 - 14 years	165.3
	M/F	5 - 14 years (blacks)	170
	M/F	5 - 14 years (whites)	162
Friedman & Goldberg (1973)	M/F	9 - 19 years	157
Webber et al (1978)	M/F	Cord blood	70
	M/F	6 months	135
	M/F	12 months	145
Freidman (1975)	M/F	0 - 5 years (Arizona)	169
	M/F	0 - 5 years (Mexico)	148

[a] Table assembled by Professor G. Christakis, University of Miami, from original sources.

Source: Conference on the Health Effects of Blood Lipids: Optimal Distributions for populations. Prev. Med. 1979; 8:612

TABLE 13

Mean cholesterol of healthy populations age 40 - 59 years in various countries (modified after Keys , 1970)

	Number	Mean cholesterol (mg.%)
Dalmatia (Croatia)	672	186
Slavonia (Yugoslavia)	699	198
West Finland	817	253
East Finland	860	265
United States	-	238
Crevalcore (Italy)	993	200
Montegiorgia (Italy)	719	200
Zutphen (Netherlands)	-	230
Crete (Greek Island)	686	202
Corfu (Greek Island)	529	198
Tanushimaru (Japan)	509	170
Ushibuka (Japan)	504	140

TABLE 14

Pool 5 and Individual Studies: Serum Cholesterol, Standardized Incidence Ratio, Risk Ratio V/(I + II), Number of Men, Person-Years of Experience, and Number of First Events by Quintile of Level

Quintile and level (mg/dl)		Study Group								
		Pool 5	ALB	CH-GAS	CH-WE	FRAM	TECUM	LA	MI-EX	MI-RR
		Standardized incidence ratio								
All	All	100	100	100	100	100	100	100	100	100
I + II	218	66	70	79	60	62	49	(42)	70	49
I	194	72	72	100	62	74	(10)	(37)	(64)	(47)
II	194-218	61	67	61	57	50	(83)	(46)	(78)	50
III	218-240	78	72	89	70	88	(56)	116	(117)	77
IV	240-268	129	129	124	99	160	145	73	(117)	96
V	268	150	177	118	159	167	242	143	(189)	194
Risk ratio V/(I + II)		2.4	2.5	1.5	2.7	2.7	4.9	( )[a]	( )	4.0
95% Confidence interval										
Low		1.9	1.7	0.9	1.7	1.7	2.0	( )	( )	3.4
High		2.9	3.8	2.4	4.6	4.0	13.1	( )	( )	7.6
Number of men at risk		8,274	1.765	1.264	1,980	2,130	1,135	1,104	283	2,551
Person years of experience		70,781	16,878	11,064	16,505	19,480	6,854	10,137	4,008	12,484
Number of first events		647	156	123	142	177	49	72	28	112

[a]( ) Based on fewer than 10 first events.

Source: The Pooling Project Research Group, 1978.

Alb = Albany civil servant
CH-GAS = Chicago peoples Gas Co.
CH-WE = Chicago Western Electric Co.
FRAM = Framingham Community
TECUM = Tecumseh Community
LA = Los Angeles
MI-Ex = Minnesota business and professional men.
MI-RR = Minnesota Railroad Workers.

TABLE 15

Epidemiological Studies on Physical Activity and CHD

Type of Study	Author	Population	Occupation	Physical Inactivity and CHD
Retrospective				
London Transport	Morris et al(1953,1966)	31,000	Drivers vs conductors	Positive association
Harvard football	Pomeroy & White (1958)		Athletes vs nonathletes	No CHD in athletes who kept active after graduation
North Dakota	Zukel et al (1959)	20,000	Farmers vs others	Positive association
Peoples Gas Co.	Stamler et al (1960)	1,500	Blue collar vs white collar	Positive association
U.S. Railroad	Taylor et al(1962,1967)	100,000	Switchmen vs clerks	Positive association
HIP of New York	Frank et al (1966)	301	Less active, intermediate, more active	Positive association
Evans Co.	Hames (1971)	5,000	Labourers vs white collar	Positive association
Danish athletes	Schnohr (1971)	307	Athletes vs nonathletes	No association(but athletes lived longer)
College Oarsmen	Prout (1972)	172	Athletes vs nonathletes	No association(but athletes lived longer)
Harvard athletes	Polednak (1972)	681	Athletes(1 or 2 letter) vs athletes(3 letters or more)	More CHD in lettermen with 3 letters or more
Prospective				
Framingham	Kannel (1967)	5,000	Active vs sedentary	Positive association
Seven countries	Keys (1970)	12,000	Active vs sedentary	No association
San Francisco	Paffenbarger et al(1970, 1975,1977)	6,351	Longshoremen - hard, medium or light work	Positive association
Goteborg	Werko (1971)	834	Active vs sedentary	Positive association
British Civil Servants	Morris et al(1973)	16,882	Active vs inactive (leisure time)	Positive association
	Chave et al (1978)	16,882		
Pathology				
England	Morris et al (1953)	3,800	Light,moderate,heavy	Positive association
De Mar	Currens & White (1961)	1	Marathon runner	Enlarged diam.of coron.art.

TABLE 16

QUALITY CONTROL OF BIOCHEMICAL VARIABLES MEASUREMENT

	No. of analyses	Mean (mg/100ml.)	S.D. (mg)	Inter-batch co-efficient of Variation%
Non-fasting	41	209.2	4.8	2.3
Serum	25	214.6	8.1	3.7
Cholesterol	22	222.8	13.8	6.2
Fasting	35	98.5	4.1	4.2
Blood glucose	38	106.3	3.6	3.4
	41	112.6	3.2	2.8

TABLE 17

RESPONSE RATE

	No.	%
Respondents	2407	91.4
Non-respondents	226	8.6
TOTAL	2633	100.0

TABLE 18

CAUSES OF NON-RESPONSE

	No.	%
Refusal	181	80.1
On study-leave	5	2.2
On special leave	9	4.0
Left employment	16	7.1
Termination of service	12	5.3
Other	3	1.3
ALL CAUSES	226	100.0

TABLE 19

AGE DISTRIBUTION OF STUDY POPULATION BY DIFFERENT INTERVIEWERS

	30 - 39 years				40 - 49 years				50 - 60 years				ALL AGES			
	Male		Female		Male		Female		Male		Female		Male		Female	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Interviewer 1	340	55.1	259	51.2	250	51.9	133	49.1	178	51.6	102	54.8	768	53.2	494	51.3
Interviewer 2	277	44.9	247	48.8	232	48.1	138	50.9	167	48.4	84	45.2	676	46.8	469	48.7
Study Population	617	100.0	506	100.0	482	100.0	271	100.0	345	100.0	186	100.0	1444	100.0	963	100.0

TABLE 20

Questionnaire results by different interviewers

Interviewers	No. Examined		Angina		PMI	
	No	%	No	%	No	%
1	1262	52.4	38	3.0	23	1.8
2	1145	47.6	36	3.1	19	1.7
Total	2407	100.0	74	3.1	42	1.7

TABLE 21

Age-Specific Means and Standard deviations of Coronary risk factors by interviewer and sex

		Interviewer 1						Interviewer 2					
		30 - 39		40 - 49		50 - 60		30 - 39		40 - 49		50 - 60	
		$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.
Height	M	172.25	6.02	172.61	5.59	170.46	6.15	171.63	5.68	173.17	5.67	170.81	5.88
	F	161.84	5.14	162.13	5.15	161.33	4.88	161.68	6.46	163.41	6.52	160.75	4.49
Weight (Kgms)	M	77.66	10.71	81.93	6.96	76.66	7.16	73.84	10.35	84.47	8.34	76.74	11.11
	F	63.33	9.55	69.77	4.55	68.25	5.92	65.31	7.66	70.61	6.84	67.85	7.77
B.M.I. (Weight/ Height^2)	M	26.22	4.16	27.51	4.79	26.35	4.21	25.04	3.14	28.86	4.59	26.24	4.14
	F	24.23	3.34	26.57	3.72	26.19	3.27	25.19	3.44	26.76	3.4	26.26	4.84
Systolic B.P. (mmHg)	M	126.34	10.64	130.78	14.79	137.19	15.35	126.78	11.77	132.37	15.61	135.49	16.59
	F	119.86	11.98	128.83	11.17	136.16	15.59	120.27	12.96	129.67	14.62	134.94	16.17
Diastolic B.P. (mmHg)	M	81.47	6.05	85.84	8.27	86.12	8.61	81.61	6.77	87.07	5.99	86.34	9.43
	F	77.16	7.41	82.31	6.41	91.91	9.77	76.94	7.01	82.71	7.42	82.79	8.47
Plasma cholesterol (mg %)	M	200.49	24.33	214.51	27.84	228.61	36.68	200.34	23.59	216.15	27.19	223.49	28.78
	F	193.74	21.12	218.46	29.43	223.92	26.91	194.87	21.74	218.16	26.14	222.02	22.44
Fasting blood sugar (mg %)	M	91.61	13.41	99.66	53.29	108.19	64.52	90.25	12.04	95.92	17.46	104.71	32.79
	F	90.55	17.92	98.27	24.95	99.25	32.51	91.06	13.31	109.44	66.65	105.27	46.68

TABLE 22

Age and sex distribution of the study population

	Age in years						Total	
	30 - 39		40 - 49		50 - 60			
	No.	% of total	No.	% of total	No.	% of total	No.	% of total
Males	617	25.6	482	20.1	345	14.4	1444	60.0
Females	506	21.0	271	11.2	186	7.7	963	40.0
Total	1123	46.6	753	31.3	531	22.1	2407	100.0

TABLE 23

Prevalence of IHD by Age & Sex

Age & Sex		N	ANGINA		N	PMI		N	ECG		N	ECG without symptoms	
			No	%		No	%		No	%		No	%
30-39	M	617	13	2.1		9	1.5		11	1.8		3	0.5
	F	506	4	0.8		2	0.4		5	1.0		2	0.4
40-49	M	482	18	3.7		10	2.1		24	5.0		14	2.9
	F	271	7	2.6		3	1.1		5	1.8		2	0.7
50-60	M	345	24	7.0		16	4.6		34	9.9		20	5.8
	F	186	8	4.3		2	1.1		12	6.5		7	3.8
Total	M	1444	55	3.8		35	2.4		59	4.8		37	2.6
	F	963	19	2.0		7	0.7		22	2.3		11	1.1

Age $P<0.001$

Sex $P<0.05$

TABLE 24

PREVALENCE (NUMBERS AND RATES PER HUNDRED) OF ELECTRO-CARDIOGRAPHIC FINDINGS - MALES

Electrocardiographic findings (and Minnesota Code)	Numbers (& rates/100)		
	30 - 39 Years	40 - 49 Years	50 - 60 Years
Q wave			
Large (1:1)	2 (0.3)	2 (0.4)	5 (1.4)
Medium (1:2)	3 (0.5)	5 (1.0)	7 (2.0)
Small (1:3)	3 (0.5)	5 (1.0)	9 (2.6)
Total (1:1-3)	8 (1.3)	12 (2.5)	21 (6.1)
Axis deviation			
Left (2:1)	3 (0.5)	9 (1.9)	11 (3.2)
Right (2:2)	0 (0)	1 (0.2)	1 (0.3)
Tall R wave (3:1)	2 (0.3)	5 (1.0)	4 (1.2)
(3:2)	1 (0.2)	3 (0.6)	3 (0.9)
(3:3)	0 (0)	2 (0.4)	2 (0.6)
ST depression			
Major (4:1)	2 (0.3)	3 (0.6)	4 (1.2)
Intermediate (4:2)	2 (0.3)	3 (0.6)	1 (0.3)
Minor (4:3)	0 (0)	2 (0.4)	3 (0.9)
Upwards sloping (4:4)	1 (0.2)	1 (0.2)	2 (0.6)
Total (4:1-4)	5 (0.8)	9 (1.9)	10 (2.9)
T wave inversion			
Major (5:1)	2 (0.3)	3 (0.6)	3 (0.9)
Intermediate (5:2)	0 (0)	2 (0.4)	4 (1.2)
Minor or flattening (5:3)	2 (0.3)	5 (1.0)	13 (3.8)
Total (5:1-3)	4 (0.6)	10 (2.1)	20 (5.8)
AV conduction defect			
3° block (6:1)			
2° block (6:2)			
1° block (6:3)	0 (0)	4 (0.8)	5 (1.4)
WPW syndrome (6:4)			
Accelerated (6:5)	0 (0)	2 (0.4)	2 (0.6)
Ventricular conduction defect			
Left BBB (7:1)	0 (0)	2 (0.4)	2 (0.6)
Other (7:2, 7:4)	0 (0)	2 (0.4)	2 (0.6)
Rhythm and rate			
Frequent premature beats (8:1)	0 (0)	4 (0.8)	3 (0.9)
Atrial fibrillation (8:3)	1 (0.2)	1 (0.2)	1 (0.3)
Rate 100/min (8:7)	2 (0.3)	4 (0.8)	6 (1.7)
Rate 50/min (8:8)			
Low QRS amplitude (9:1)	4 (0.6)	6 (1.2)	6 (1.7)
STUDY POPULATION	617	482	345

TABLE 25

PREVALENCE (NUMBERS AND RATES PER HUNDRED) OF ELECTRO-CARDIOGRAPHIC FINDINGS - FEMALES

Electrocardiographic findings (and Minnesota Code)	Numbers (& rates/100)		
	30 - 39 Years	40 - 49 Years	50 - 60 Years
Q wave			
Large (1:1)	1 (0.2)	1 (0.4)	1 (0.5)
Medium (1:2)	2 (0.4)	2 (0.7)	3 (1.6)
Small (1:3)	3 (0.6)	2 (0.7)	2 (1.1)
Total (1:1-3)	6 (1.2)	5 (1.8)	6 (3.2)
Axis deviation			
Left (2:1)	1 (0.2)	2 (0:7)	3 (1:6)
Right	0 (0)	0 (0)	0 (0)
Tall R wave (3:1)	1 (0.2)	0 (0)	1 (0.5)
(3:2)	0 (0)	0 (0)	1 (0.5)
ST depression			
Major (4:1)	1 (0.2)	1 (0.4)	1 (0.5)
Intermediate (4:2)	0 (0)	1 (0.4)	1 (0.5)
Minor (4:3)	1 (0.2)	0 (0)	2 (1.1)
Upwards sloping (4:4)	0 (0)	0 (0)	0 (0)
Total (4:1-4)	2 (0.4)	2 (0.7)	4 (2.1)
T wave inversion			
Major (5:1)	0 (0)	1 (0.4)	1 (0.5)
Intermediate (5:2)	0 (0)	0 (0)	2 (1.1)
Minor or flattening (5:3)	0 (0)	2 (0.7)	2 (1.1)
Total (5:1-3)	0 0	3 (1.1)	5 (2.7)
AV conduction defect			
3° block (6:1)			
2° block (6:2)			
1° block (6:3)			
WPW syndrome (6:4)			
Accelerated (6:5)	0 (0)	0 (0)	1 (0.5)
Ventricular conduction defect			
Left BBB(7:1)	1 (0.2)	1 (0.4)	2 (1.1)
Other (7:2, 7:4)			
Rhythm and rate			
Frequent premature beats (8:1)	0 (0)	0 (0)	2 (1.1)
Atrial fibrillation (8:3)	0 (0)	0 (0)	0 (0)
Rate 100/min (8:7)	0 (0)	1 (0.4)	1 (0.5)
Rate 50/min (8:8)			
Low QRS amplitude (9:1)	2 (0.4)	3 (1.1)	3 (1.6)
STUDY POPULATION	506	271	186

TABLE 26

Ischaemic-type ECG changes with and without symptoms

		N	ECGs with symptoms		ECGs without symptoms	
			No	%	No	%
30-39	M	617	8	1.2	3	0.5
	F	506	3	0.6	2	0.4
40-49	M	482	10	2.1	14	2.9
	F	271	3	1.1	2	0.7
50-60	M	345	14	4.1	20	5.8
	F	186	5	2.7	7	3.8
Total	M	1444	32	2.2	37	2.6
	F	963	11	1.2	11	1.1

TABLE 27

PREVALENCE OF ANGINA AND ITS RELATION TO ISCHAEMIC-TYPE ELECTROCARDIOGRAPHIC FINDINGS*

Angina	Age & Sex Categories						All Ages	
	30 - 39 years		40 - 49 years		50 - 60 years			
	Males	Females	Males	Females	Males	Females	Males	Females
Prevalence Rate %	2.1	0.8	3.7	2.6	6.9	4.3	3.8	1.9
** % with Positive E.C.G.	30.8	25.0	33.3	28.6	33.3	37.5	32.7	31.6

* Minnesota Codes 1:1-3, 4:1-3, 5:1-3 or 7:1.

** Age-adjusted % Males = 32.23
Females = 28.44

TABLE 28

Means and Standard Deviations of Variables in age and sex groups - the entire population

Variable	Sex	Age Categories					
		30 - 39		40 - 49		50 - 60	
		$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.
Height	M	171.97	5.87	172.39	5.61	170.63	6.01
	F	161.85	5.43	162.27	5.33	161.06	4.71
Weight	M	75.94	14.22	83.16	7.78	76.71	12.51
	F	68.21	8.47	70.19	12.5	68.07	8.57
BMI (Weight/Height^2)	M	25.69	3.38	28.16	4.67	26.31	3.73
	F	26.17	3.11	26.67	4.65	26.23	4.08
Systolic B.P.	M	126.54	11.16	131.54	15.19	136.37	16.96
	F	120.07	12.46	129.26	13.03	135.16	15.86
Diastolic B.P.	M	81.53	6.38	86.43	9.87	87.23	9.01
	F	77.05	7.21	82.52	6.94	86.79	9.39
Plasma Cholesterol	M	200.43	23.98	215.29	27.52	225.64	33.19
	F	194.29	21.42	218.31	28.75	223.06	24.95
Fasting blood glucose	M	91.01	12.82	97.86	40.25	106.51	58.14
	F	90.81	15.83	104.45	57.54	101.97	39.54

TABLE 29

DISTRIBUTION OF FAT USED FOR COOKING BY AGE AND SEX

		30-39 years		40-49 years		50-60 years		N	
		Males	Females	Males	Females	Males	Females	Males	Females
Animal Fat	No.	104	66	132	69	113	77	349	212
	%	16.9	13.0	27.4	25.5	32.8	41.4	24.2	22.0
Vegetable Fat	No.	308	225	204	98	157	77	669	400
	%	49.9	44.5	42.3	36.2	45.5	41.4	46.3	41.5
Both	No.	205	215	146	104	75	32	426	351
	%	33.2	42.5	30.3	38.4	21.7	17.2	29.5	36.4
T O T A L	No.	617	506	482	271	345	186	1444	964
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 30

DISTRIBUTION OF FAT USED FOR FRYING BY AGE AND SEX

		30-39 years		40-49 years		50-60 years		N	
		Males	Females	Males	Females	Males	Females	Males	Females
Animal Fat	No.	20	12	23	16	26	21	69	49
	%	3.2	2.4	4.8	5.9	7.5	11.3	4.8	5.1
Vegetable Fat	No.	456	379	337	152	227	123	1020	672
	%	73.9	78.5	69.9	56.1	65.8	66.1	70.6	69.8
Both	No.	141	97	122	103	92	42	355	242
	%	22.9	19.2	25.3	38.0	26.7	22.6	24.6	25.1
T O T A L	No.	617	506	482	271	345	186	1444	963
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 31

DISTRIBUTION OF FAT USED FOR SPREADING BY AGE AND SEX

		30-39 years		40-49 years		50-60 years		N	
		Males	Females	Males	Females	Males	Females	Males	Females
Animal Fat	No.	24	19	32	20	17	11	73	50
	%	3.9	3.8	6.6	7.4	4.9	5.9	5.1	5.2
Vegetable Fat	No.	454	333	304	135	212	92	970	560
	%	73.6	65.8	63.1	49.8	61.4	49.5	67.2	58.2
Both	No.	139	154	146	116	116	83	401	83
	%	22.5	30.4	30.3	42.8	33.6	44.6	17.8	44.6
T O T A L	No.	617	506	482	271	345	186	1444	963
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 32

DISTRIBUTION OF BREAD EATEN BY AGE AND SEX

		30-39 years		40-49 years		50-60 years		N	
		Males	Females	Males	Females	Males	Females	Males	Females
White Bread	No.	283	244	240	141	164	104	687	489
	%	45.8	48.2	49.8	52.1	47.5	55.9	47.6	50.8
Brown Bread	No.	64	23	61	44	53	32	178	99
	%	10.4	4.6	12.6	16.2	15.4	17.2	12.3	10.3
Both	No.	270	239	181	86	128	50	579	375
	%	43.8	47.2	37.6	31.7	37.1	26.9	40.1	38.9
Total	No.	617	506	482	271	345	186	1444	963
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 33

DISTRIBUTION OF MEAT CONSUMPTION BY AGE AND SEX

		30-39 years		40-49 years		50-60 years		N	
		Males	Females	Males	Females	Males	Females	Males	Females
Daily	No.	156	284	164	118	127	79	447	481
	%	25.3	56.1	34.0	43.5	36.8	42.5	31.0	49.9
Three times weekly	No.	367	187	220	113	139	88	726	388
	%	59.5	36.9	45.6	41.7	40.3	47.3	50.3	40.3
Weekly	No.	94	35	98	40	79	19	271	94
	%	15.2	6.9	20.3	14.8	22.9	10.2	18.8	9.8
Total	No.	617	506	482	271	345	186	1444	963
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 34

DISTRIBUTION OF GREEN BEANS INTAKE BY AGE AND SEX

		30 - 39		40 - 49		50 - 60		N	
		Males	Females	Males	Females	Males	Females	Males	Females
Daily	No.	15	20	12	6	7	3	34	29
	%	2.4	4.0	2.5	2.2	2.0	1.6	2.4	3.0
Three times weekly	No.	71	44	69	25	53	9	193	78
	%	11.5	8.7	14.3	9.2	15.4	4.8	13.4	8.1
Weekly	No.	531	442	401	240	285	174	1217	856
	%	86.1	87.4	83.2	88.6	82.6	93.5	84.3	88.9
Total	No.	617	506	482	271	345	186	1444	963
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 35

DISTRIBUTION OF DRY BEANS INTAKE BY AGE AND SEX

		30 - 39		40 - 49		50 - 60		N	
		Males	Females	Males	Females	Males	Females	Males	Females
Daily	No.	9	18	5	3	6	4	20	25
	%	1.5	3.6	1.0	1.1	1.7	2.2	1.4	2.6
Three times weekly	No.	32	23	38	28	32	4	102	55
	%	5.2	4.5	7.9	10.3	9.3	2.2	7.1	5.7
Weekly	No.	576	465	439	240	307	178	1322	883
	%	93.4	91.9	91.1	88.6	89.0	95.7	91.6	91.7
Total	No.	617	506	482	271	345	186	1444	963
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 36

DISTRIBUTION OF CITRUS FRUIT INTAKE BY AGE AND SEX

		30 - 39		40 - 49		50 - 60		N	
		Males	Females	Males	Females	Males	Females	Males	Females
Daily	No.	211	344	183	132	131	78	525	554
	%	34.2	68.0	38.0	48.7	38.0	41.9	36.4	57.5
Three times weekly	No.	292	123	223	110	156	89	671	322
	%	47.3	24.3	46.3	40.6	45.2	47.8	46.5	33.4
Weekly	No.	114	39	76	29	58	19	248	87
	%	18.5	7.7	15.8	10.7	16.8	10.2	17.2	9.0
Total	No.	617	506	482	271	345	186	1444	963
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 37

DISTRIBUTION OF OTHER FRUIT (APPLES AND BANANAS) INTAKE BY AGE AND SEX

		30 - 39		40 - 49		50 - 60		N	
		Males	Females	Males	Females	Males	Females	Males	Females
Daily	No.	188	309	145	96	124	46	457	451
	%	30.5	61.1	30.1	35.4	35.9	24.7	31.6	46.8
Three times weekly	No.	259	146	206	112	129	87	594	345
	%	42.0	28.9	42.7	41.3	37.4	46.8	41.1	35.8
Weekly	No.	170	51	131	63	92	53	393	167
	%	27.6	10.1	27.2	23.2	26.7	28.5	27.2	17.3
Total	No.	617	506	482	271	345	186	1444	963
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 38

DIET SCORING SCALE

What kind of fat do you use?	Animal	Vegetable	Both
For Cooking	1.00	.33	.66
For Frying	1.00	.33	.66
For Spreading	1.00	.33	.66
What kind of bread do you eat?	**White**	**Brown**	**Both**
	1.00	.33	.66
How often do you eat?	**Daily**	**3 times a week**	**Weekly**
Meat	1.00	.66	.33
Dry Beans	.33	.66	1.00
Green Beans	.33	.66	1.00
Citrus Fruit	.33	.66	1.00
Other Fruit (apples and bananas)	.33	.66	1.00

TABLE 39

Prevalence of Angina and PMI and Diet

Age and Sex		ANGINA						PMI					
		N	DPF <0.66 No.	%	N	DPF ≥0.66 No.	%	N	DPF <0.66 No.	%	N	DPF ≥0.66 No.	%
30 - 39	M	207	5	2.4	410	8	2.0	207	3	1.4	410	6	1.5
	F	230	3	1.3	276	1	0.4	230	0	0	276	2	0.7
40 - 49	M	138	4	2.9	344	14	4.1	138	4	2.9	344	6	1.7
	F	68	0	0	203	7	3.4	68	2	2.9	203	1	0.5
50 - 60	M	105	5	4.8	240	19	7.9	105	6	5.7	240	10	4.2
	F	29	1	3.4	157	7	4.5	29	0	0	157	2	1.3
Total	M	450	14	3.1	994	41	4.1	450	13	2.9	994	22	2.2
	F	327	4	1.2	636	15	2.4	327	2	0.6	363	5	0.8
Age-adjusted	M			3.1			4.1			2.9			2.2
	F			1.3			2.0			0.8			0.8

Significance of diet controlling for age and sex P >0.1 P >0.1

TABLE 40

Prevalence of ischaemic-type ECG changes and ischaemic-type ECG changes without symptoms and Diet

Age and Sex		Ischaemic-type ECG changes						Ischaemic-type ECG changes without symptoms					
		N	DPF <0.66 No.	%	N	DPF ≥0.66 No.	%	N	DPF <0.66 No.	%	N	DPF ≥0.66 No.	%
30 - 39	M	207	3	1.4	410	8	2.0	207	0	0	410	3	0.7
	F	230	0	0	276	5	1.8	230	0	0	276	2	0.7
40 - 49	M	138	4	2.9	344	20	5.8	138	1	0.7	344	13	3.8
	F	68	1	1.5	203	4	2.0	68	0	0	203	2	1.0
50 - 60	M	105	12	11.4	240	22	9.2	105	5	4.8	240	15	6.3
	F	29	0	0	157	12	7.6	29	0	0	157	7	4.5
Total	M	450	19	4.2	994	50	5.0	450	6	1.3	994	31	3.1
	F	327	1	0.3	636	21	3.3	327	0	0	636	11	1.7
Age-adjusted	M			4.3			4.9			1.4			3.1
	F			0.4			2.9			0.0			1.5
Significance of diet controlling for age and sex				P>0.1						P <0.05			

TABLE 41

Prevalence of angina and PMI and Fat

Age and Sex		ANGINA						PMI					
		N	FPF < 0.66 No.	%	N	FPF ≥ 0.66 No.	%	N	FPF < 0.66 No.	%	N	FPF ≥ 0.66 No.	%
30 - 39	M	504	10	2.0	113	3	2.7	504	6	1.2	113	3	2.7
	F	397	4	1.0	109	0	0	397	1	0.3	109	1	0.9
40 - 49	M	329	13	4.0	153	5	3.3	329	8	2.4	153	2	1.3
	F	151	3	2.0	120	4	3.3	151	1	0.7	120	2	1.7
50 - 60	M	216	12	5.6	129	12	9.3	216	10	4.6	129	6	4.7
	F	91	5	5.5	95	3	3.2	91	0	0	95	2	2.1
Total	M	1049	35	3.3	395	20	5.1	1049	24	2.3	395	11	2.8
	F	639	12	1.9	324	7	2.2	639	2	0.3	324	5	1.5
Age-adjusted	M			3.5			4.5			2.4			2.7
	F			2.1			1.5			0.3			1.4
Significance of fat controlling for age and sex				P > 0.1						P > 0.1			

TABLE 42

Prevalence of ischaemic-type ECG changes and ischaemic-type ECG changes without symptoms and fat

Age and sex		Ischaemic-type ECG changes						Ischaemic-type ECG changes without symptoms					
		N	FPF < 0.66 No.	%	N	FPF ≥ 0.66 No.	%	N	FPF < 0.66 No.	%	N	FPF ≥ 0.66 No.	%
30 - 39	M	504	8	1.6	113	3	2.7	504	3	0.6	113	0	0
	F	397	3	0.8	109	2	1.8	397	1	0.3	109	1	0.9
40 - 49	M	329	12	3.6	153	12	7.8	329	4	1.2	153	10	6.5
	F	151	2	1.3	120	3	2.5	151	1	0.7	120	1	0.8
50 - 60	M	216	22	10.2	129	12	9.3	216	13	6.0	129	7	5.4
	F	91	2	2.2	95	10	10.5	91	1	1.1	95	6	6.3
Total	M	1049	42	4.0	395	27	6.8	1049	20	1.9	395	17	4.3
	F	639	7	1.1	324	15	4.6	639	3	0.5	424	8	2.5
Age-adjusted	M			4.3			5.9			2.1			3.5
	F			1.2			3.7			0.6			1.9
Significance of fat controlling for age and sex				$P < 0.05$						$P < 0.05$			

TABLE 43

Prevalence of Angina & type of bread eaten

Age & Sex		White bread			Brown bread			Both		
		N	ANGINA No	%	N	ANGINA No	%	N	ANGINA No	%
30-39	M	283	9	3.2	64	0	0	270	4	1.5
	F	244	1	0.4	23	0	0	239	3	1.3
40-49	M	240	12	5.0	61	2	3.3	181	4	2.2
	F	141	5	3.5	44	2	4.5	86	0	0
50-60	M	164	15	9.1	53	4	7.5	128	5	3.9
	F	104	4	3.8	32	0	0	50	4	8.0
Total	M	687	36	5.2	178	6	3.3	579	13	2.2
	F	489	10	2.1	99	2	2.0	375	7	1.8
Age-adjusted	M			5.2			2.9			2.3
	F			1.9			1.3			2.2

Significance of type of bread controlling for age and sex $0.1 > P > 0.05$

TABLE 44

Prevalence of Possible Myocardial Infarction & type of bread eaten

Age & Sex		White bread			Brown bread			Both		
		N	PMI No	PMI %	N	PMI No	PMI %	N	PMI No	PMI %
30-39	M	283	7	2.5	64	1	1.6	270	1	0.4
	F	244	2	0.8	23	0	0	239	0	0
40-49	M	240	6	2.5	61	2	3.3	181	2	1.1
	F	141	2	1.4	44	0	0	86	1	1.2
50-60	M	164	9	5.5	53	2	3.8	128	5	3.9
	F	104	2	1.9	32	0	0	50	0	0
Total	M	687	22	3.2	178	5	2.8	579	8	1.4
	F	489	6	1.2	99	0	0	375	1	0.3
Age-adjusted	M			3.2			2.7			1.5
	F			1.2			0.00			0.3

Significance of type of bread controlling for age and sex $P<0.05$

TABLE 45

Prevalence of ischaemic type ECG & type of bread eaten

Age & Sex		White bread			Brown bread			Both		
		N	ECG No	ECG %	N	ECG No	ECG %	N	ECG No	ECG %
30-39	M	283	9	3.2	64	1	1.6	270	1	0.4
	F	244	5	2.0	23	0	0	239	0	0
40-49	M	240	18	7.5	61	1	1.6	181	5	2.8
	F	141	4	2.8	44	0	0	86	1	1.2
50-60	M	164	22	13.4	53	1	1.9	128	11	8.6
	F	104	7	6.7	32	0	0	50	5	10.0
Total	M	687	49	7.1	178	3	1.7	579	17	2.9
	F	489	16	3.3	99	0	0	375	6	1.6
Age-adjusted	M			7.1			1.7			3.2
	F			3.1			0.0			2.3

Significance of type of bread controlling for age and sex $P < 0.001$

TABLE 46

Prevalence of ischaemic type ECG without symptoms and type of bread eaten

Age & Sex		White bread			Brown bread			Both		
		N	ECG without symptoms No	%	N	ECG without symptoms No	%	N	ECG without symptoms No	%
30-39	M	283	2	0.7	64	1	1.6	270	0	0
	F	244	2	0.8	23	0	0	239	0	0
40-49	M	240	10	4.2	61	0	0	181	4	2.2
	F	141	2	1.4	44	0	0	86	0	0
50-60	M	164	14	8.5	53	0	0	128	6	4.7
	F	104	4	3.8	32	0	0	50	3	6.0
Total	M	687	26	3.8	178	1	0.6	579	10	1.7
	F	489	8	1.6	99	0	0	375	3	0.8
Age-adjusted	M			3.7			0.7			1.8
	F			1.5			0.0			1.2

Significance of type of bread controlling for age and sex $P<0.01$

TABLE 47

Age-specific Means & Standard Deviations of Coronary Risk Factors by DPF & Sex

		Males						Females					
		30-39		40-49		50-60		30-39		40-49		50-60	
		$\bar{x}$	S.D.	$\bar{x}$	S.D.	$\bar{x}$	S.D.	$\bar{x}$	S.D.	$\bar{x}$	S.D.	$\bar{x}$	S.D.
Height (Cms)	DPF ≥ 0.66	172.17	5.79	172.68	5.61	170.86	5.96	161.59	5.38	161.79	5.17	160.85	4.67
	DPF < 0.66	171.57	6.03	171.68	5.53	170.11	6.13	162.16	5.47	163.71	5.61	162.21	4.82
Weight (Kgms)	DPF ≥ 0.66	77.52	9.99	81.83	13.15	77.42	10.41	68.05	5.56	69.21	10.16	68.25	8.74
	DPF < 0.66	72.82	10.19	81.47	8.94	75.06	9.99	68.41	7.03	73.16	8.55	67.13	7.68
BMI (Weight/ Height^2)	DPF ≥ 0.66	26.18	3.49	27.63	4.23	26.49	4.08	26.21	4.69	26.46	3.91	26.37	3.17
	DPF < 0.66	24.72	4.16	27.47	4.05	25.88	3.75	26.11	4.79	27.31	4.38	25.49	4.42
Sytolic B.P. (MM Hg)	DPF ≥ 0.66	126.64	11.13	132.34	15.75	137.18	16.91	121.41	14.22	128.91	12.77	137.17	16.62
	DPF < 0.66	126.32	11.24	129.56	13.58	134.52	13.45	118.45	9.75	130.29	13.82	130.69	9.23
Diastolic B.P. (mm Hg)	DPF ≥ 0.66	81.59	6.42	87.84	8.79	86.21	9.02	77.63	7.59	82.49	7.25	88.85	4.55
	DPF < 0.66	81.41	6.31	82.91	7.74	86.28	9.01	76.37	6.68	82.57	5.95	82.07	6.75
Plasma Choles-terol (mg%)	DPF ≥ 0.66	202.14	24.64	216.71	27.34	227.01	29.24	196.51	22.61	217.41	27.52	223.23	25.16
	DPF < 0.66	197.03	22.31	211.78	27.74	222.52	40.79	191.65	19.61	221.03	28.47	222.17	24.15
Fasting Blood Glucose	DPF ≥ 0.66	91.13	13.19	98.52	28.15	103.73	33.56	92.23	19.36	105.08	44.92	103.29	42.63
	DPF < 0.66	90.74	12.05	101.18	36.96	112.85	29.38	89.11	16.85	102.55	25.23	94.79	11.77

TABLE 48

Age-Specific Means and Standard Deviation by FPF and Sex

		Males						Females					
		30 - 39		40 - 49		50 - 60		30 - 39		40 - 49		50 - 60	
		$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.
Height	FPF ≥0.66	171.46	5.97	172.75	5.04	171.53	5.68	161.51	5.14	161.68	5.38	161.04	4.82
	FPF <0.66	172.08	5.85	172.46	5.85	170.08	6.15	161.94	5.51	162.74	5.96	161.09	4.62
Weight (Kgms)	FPF ≥0.66	79.19	6.82	84.29	4.91	80.14	14.59	68.41	6.13	69.69	9.84	68.56	8.28
	FPF <0.66	75.22	3.92	82.63	6.16	74.66	10.59	67.47	5.32	70.59	10.31	67.61	8.86
BMI (Weight/ Height^2)	FPF ≥0.66	26.95	3.11	28.56	4.92	27.22	4.56	26.13	5.12	26.67	3.71	26.07	3.31
	FPF <0.66	25.41	4.58	27.97	4.27	25.75	3.02	24.18	3.41	26.68	3.29	25.41	4.83
Systolic B.P. (mmHg)	FPF ≥0.66	127.31	11.81	132.42	14.36	137.59	14.84	122.02	15.49	128.63	13.11	136.75	17.06
	FPF <0.66	126.36	11.01	131.14	15.57	135.65	16.59	119.53	11.46	129.76	12.99	135.58	14.68
Diastolic B.P. (mmHg)	FPF ≥0.66	81.64	5.99	86.61	8.51	86.68	7.52	78.58	8.25	82.81	7.18	92.81	8.43
	FPF <0.66	81.51	6.47	86.35	5.05	85.02	9.79	76.63	6.85	82.28	6.75	83.01	8.79
Plasma cholesterol (mg %)	FPF ≥0.66	203.47	28.82	219.85	31.14	230.41	31.07	200.73	25.43	218.81	22.59	224.43	24.81
	FPF <0.66	199.74	22.73	213.17	25.43	222.81	34.14	192.53	19.85	220.29	31.19	223.72	25.22
Fasting blood sugar	FPF ≥0.66	89.88	9.37	97.38	20.86	105.31	37.35	95.29	27.57	101.96	27.94	101.54	33.58
	FPF <0.66	91.25	13.46	98.08	46.63	107.23	57.65	89.57	10.24	106.43	41.61	102.43	45.12

TABLE 49

Age Specific Means and Standard Deviations of Coronary Risk Factors by Bread and Sex

		White bread						Brown bread						Both					
		30 - 39		40 - 49		50 - 60		30 - 39		40 - 49		50 - 60		30 - 39		40 - 49		50 - 60	
		$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.
Height	M	171.73	6.15	172.61	5.43	170.79	5.75	172.03	4.17	170.61	6.21	169.72	6.36	172.21	5.93	172.72	5.54	170.81	6.22
	F	161.76	5.27	162.19	5.44	161.32	4.89	163.96	6.28	162.25	6.15	161.38	3.88	161.74	5.48	162.42	4.74	160.36	4.81
Weight (Kgms)	M	75.59	4.12	83.94	9.84	78.08	11.18	76.58	7.87	78.31	7.97	77.39	8.81	73.81	10.07	81.11	8.47	74.66	10.55
	F	69.26	5.89	69.16	4.65	67.62	8.82	62.17	8.39	69.70	6.73	69.19	9.56	67.72	5.12	72.14	11.72	68.32	7.42
BMI (Weight/ Height^2)	M	25.66	3.23	28.31	3.79	26.74	3.41	24.34	3.45	27.88	4.19	26.78	4.59	24.87	3.07	27.41	4.06	25.55	3.01
	F	26.59	3.28	26.31	4.03	26.98	3.19	23.16	3.15	26.51	3.59	26.52	3.13	26.04	2.5	27.36	4.44	26.58	2.77
Systolic B.P. (mmHg)	M	128.15	11.5	133.18	16.05	136.92	16.02	125.05	10.34	128.93	14.47	134.91	12.73	126.59	10.91	130.28	14.12	135.29	17.13
	F	119.72	12.12	129.04	14.37	136.45	16.56	117.83	5.99	128.41	9.07	132.66	10.39	118.64	13.25	130.06	12.52	137.79	17.15
Diastolic B.P. (mmHg)	M	81.64	6.66	86.34	9.03	86.07	8.75	81.33	5.36	83.15	7.48	85.09	7.10	81.46	6.32	87.71	7.88	86.91	9.98
	F	77.19	7.55	82.39	6.99	91.54	9.04	77.61	4.23	81.14	5.59	80.63	6.06	76.87	7.11	83.43	7.41	84.59	9.36
Plasma cholesterol (mg %)	M	200.99	25.02	215.82	26.93	229.73	37.92	203.61	23.73	210.69	34.09	221.12	26.69	199.09	22.92	216.18	25.85	222.29	28.37
	F	194.19	21.83	217.68	23.54	230.01	25.73	186.87	20.99	217.43	29.79	213.51	20.67	195.12	20.98	219.80	32.89	223.67	24.95
Fasting blood sugar (mg %)	M	91.23	13.99	99.26	54.43	109.79	77.48	92.31	16.00	97.91	22.69	106.13	37.76	90.46	10.51	96.09	15.27	102.46	28.94
	F	90.48	13.65	96.15	20.14	97.90	20.18	87.87	9.21	95.36	22.93	100.09	26.36	91.43	18.22	109.41	43.82	111.64	43.08

TABLE 50

Age-adjusted Percents of Coronary Risk Factors by Diet & Sex

	MALES		FEMALES	
	FPF ⩾ 0.66	FPF < 0.66	FPF ⩾ 0.66	FPF < 0.66
Height Percent ⩾ 176 cms (Males) Percent ⩾ 166 cms (Females)	22.5	19.3	17.5	23.3
BMI (Weight/Height^2) Percent ⩾ 28	23.2	18.8	19.3	15.6
Blood pressure Percent Normotensive Percent Borderline Hypertensive Percent Hypertensive	 65.7 24.2 10.1	 69.1 21.6 9.3	 81.1 11.2 7.7	 85.4 9.7 4.9
Plasma cholesterol Percent ⩾ 220 mg%	37.7	27.0	33.2	22.9
Fasting Blood glucose Percent ⩾ 120 mg%	4.2	4.9	5.1	2.7
Percent smokers Percent ex-smokers	47.1 14.1	45.5 15.1	16.3 3.8	11.6 3.6
Exercise in leisure time Percent doing vigorous exercise	33.1	41.8	35.5	40.4
Behaviour Pattern Percent Type A	52.1	44.9	49.2	53.5

TABLE 51

Age-adjusted Percents of Coronary Risk Factors by Fat Intake & Sex

	MALES		FEMALES	
	FPF ≥ 0.66	FPF < 0.66	FPF ≥ 0.66	FPF < 0.66
Height Percent ≥ 176 cms (Males) Percent ≥ 166 cms (Females)	20.8	21.4	16.2	20.1
BMI (Weight/Height^2) Percent ≥ 28	27.2	19.3	21.1	17.2
Blood Pressure Percent Normotensive Percent Borderline Hypertensive Percent Hypertensive	 64.9 24.5 10.6	 67.4 23.4 9.2	 81.8 9.9 8.3	 82.8 10.9 6.3
Plasma cholesterol Percent ≥ 220 mg%	42.6	32.5	36.7	30.7
Fasting Blood glucose Percent ≥ 120 mg%	5.1	3.1	4.3	5.1
Percent smokers Percent ex-smokers	46.5 12.3	41.7 15.4	17.9 4.3	13.4 3.8
Exercise in leisure time Percent doing exercise	30.8	37.1	37.7	37.0
Behaviour Pattern Percent Type A	53.7	48.5	47.2	52.5

TABLE 52

Age-adjusted Percents of Coronary Risk Factors by Type of Bread & Sex

	Males			Females		
	White Bread	Brown Bread	Both	White Bread	Brown Bread	Both
Height Percent ≥176 cms (Males) Percent ≥166 cms (Females)	21.3	18.4	22.8	18.9	22.2	17.7
BMI (Weight/Height^2) Percent ≥28	25.1	19.4	19.1	16.4	20.8	19.8
Blood Pressure Percent Normotensive Percent Borderline Hypertensive Percent Hypertensive	64.0 24.9 11.1	67.6 26.6 5.8	69.5 20.7 9.8	81.5 10.4 8.1	90.2 8.6 1.2	82.9 10.3 6.8
Plasma cholesterol Percent ≥220 mg%	37.6	34.5	33.1	34.3	27.8	31.6
Fasting Blood Glucose Percent ≥120 mg%	3.8	6.1	4.5	2.9	4.9	6.2
Smoking Percent smokers Percent ex smokers Percent never smoked	50.1 13.1 36.8	49.4 9.9 40.7	41.4 17.5 41.1	15.1 4.0 80.9	9.6 4.1 86.3	15.3 3.9 80.8
Exercise in leisure time Percent doing exercise Percent not doing exercixe	32.9 67.1	41.6 58.4	37.2 62.8	35.8 64.2	39.6 60.4	36.6 63.4
Behaviour Pattern Percent Type A	50.7	54.4	47.9	52.8	48.2	51.7

TABLE 53

PREVALENCE OF PLASMA CHOLESTEROL

AGE & SEX		N	PLASMA CHOLESTEROL LEVEL							
			< 200 mg%		200-219 mg %		220-239 mg%		> 240 mg%	
			No	%	No	%	No	%	No	%
30-39	M	617	340	55.11	164	26.58	67	10.86	46	7.45
	F	506	314	62.05	109	21.54	76	15.02	7	1.38
40-49	M	482	146	30.29	133	27.59	110	22.82	93	19.29
	F	271	77	28.41	78	28.78	72	26.57	44	16.24
50-60	M	345	75	21.74	74	21.45	87	25.22	109	31.59
	F	186	42	22.58	39	20.97	57	30.64	48	25.81
Total	M	1444	561	38.85	371	25.69	264	18.28	248	17.17
	F	963	433	44.96	226	23.47	205	21.29	99	10.28

TABLE 54

Prevalence of Angina & Plasma Cholesterol Level

Age & Sex		Plasma Cholesterol < 200 mg%			Plasma Cholesterol 200-219 mg%			Plasma Cholesterol 220-239 mg%			Plasma Cholesterol ≥240 mg%		
		N	ANGINA No	%	N	ANGINA No	%	N	ANGINA No	%	N	ANGINA No	%
30-39	M	340	3	0.9	164	3	1.8	67	4	5.9	46	3	6.5
	F	314	1	0.3	109	1	0.9	76	2	2.6	7	0	0
40-49	M	146	1	0.7	133	5	3.7	110	3	2.7	93	9	9.7
	F	77	1	1.3	78	2	2.5	72	1	1.4	44	3	6.8
50-60	M	75	1	1.3	74	3	4.1	87	5	5.7	109	15	13.7
	F	42	1	2.4	39	0	0	57	5	8.8	48	2	4.2
Total	M	561	5	0.9	371	11	2.9	264	12	4.5	248	27	10.9
	F	433	3	0.7	226	3	1.3	205	8	3.9	99	5	5.1
Age-adjusted	M			0.9			2.9			4.8			9.3
	F			0.9			1.2			3.4			2.7

Significance of plasma cholesterol level, testing for trend, controlling for age and sex $P < .001$

TABLE 55

Prevalence of Possible Myocardial Infarction & Plasma Cholesterol Level

Age & Sex		Plasma Cholesterol < 200 mg%			Plasma Cholesterol 200-219 mg%			Plasma Cholesterol 220-239 mg%			Plasma Cholesterol ⩾240 mg%		
		N	PMI No	PMI %	N	PMI No	PMI %	N	PMI No	PMI %	N	PMI No	PMI %
30-39	M	340	1	0.3	164	2	1.2	67	3	4.5	46	3	6.5
	F	314	0	0	109	0	0	76	1	1.3	7	1	14.3
40-49	M	146	1	0.7	133	1	0.8	110	3	2.7	93	5	5.4
	F	77	0	0	78	0	0	72	1	1.4	44	2	4.5
50-60	M	75	0	0	74	0	0	87	3	3.4	109	13	11.9
	F	42	0	0	39	1	2.6	57	1	1.8	48	0	0
Total	M	561	2	0.4	371	3	0.8	264	9	3.4	248	21	8.5
	F	433	0	0	226	1	0.4	205	3	1.5	99	3	3.0
Age-adjusted	M			0.4			0.8			3.7			7.4
	F			0.0			0.5			1.4			8.8

Significance of plasma cholesterol level, testinf for trend, controlling for age and sex $P < 0.001$

TABLE 56

Prevalence of Ischaemic type ECG & Plasma Cholesterol Level

Age & Sex		Plasma Cholesterol < 200 mg%			Plasma Cholesterol 200-219 mg%			Plasma Cholesterol 220-239 mg%			Plasma Cholesterol ≥ 240 mg%		
		N	ECG No	ECG %	N	ECG No	ECG %	N	ECG No	ECG %	N	ECG No	ECG %
30-39	M	340	2	0.5	164	1	0.6	67	3	4.4	46	5	10.8
	F	314	0	0	109	2	1.8	76	2	2.6	7	1	14.2
40-49	M	146	3	2.0	133	2	1.5	110	8	7.2	93	11	11.8
	F	77	0	0	78	0	0	72	3	4.1	44	2	4.5
50-60	M	75	2	2.6	74	5	6.7	87	5	5.7	109	22	20.2
	F	42	0	0	39	2	5.1	57	6	10.5	48	4	8.3
Total	M	561	7	1.2	271	8	2.1	264	16	6.1	248	38	15.3
	F	433	0	0	226	4	1.7	205	11	5.3	99	7	7.1
Age-adjusted	M			1.5			2.4			5.6			13.4
	F			0.0			1.9			4.5			10.3

Significance of plasma cholesterol level, testing for trend, controlling for age and sex $P < 0.001$

TABLE 57

Prevalence of Ischaemic type ECG without Symptoms & Plasma Cholesterol Level

Age & Sex		Plasma Cholesterol < 200 mg%			Plasma Cholesterol 200-219 mg%			Plasma Cholesterol 220-239 mg%			Plasma Cholesterol ⩾ 240 mg%		
		N	ECG without symptoms No	%	N	ECG without symptoms No	%	N	ECG without symptoms No	%	N	ECG without symptoms No	%
30-39	M	340	0	0	164	0	0	67	1	1.5	46	2	4.3
	F	314	0	0	109	2	1.8	76	0	0	7	0	0
40-49	M	146	2	1.3	133	2	1.5	110	4	3.6	93	6	6.4
	F	77	0	0	78	0	0	72	2	2.8	44	0	0
50-60	M	75	2	2.6	74	5	6.7	87	4	4.6	109	9	8.2
	F	42	0	0	39	1	2.5	57	3	5.2	48	3	6.2
Total	M	561	4	0.7	371	7	1.9	264	9	3.4	248	17	6.8
	F	433	0	0	226	3	1.3	205	5	2.4	99	3	3.0
Age-adjusted	M			1.1			2.1			2.9			5.9
	F			0.0			1.4			1.8			1.2

Significance of plasma cholesterol level, testing for trend, controlling for age and sex $P < 0.01$

TABLE 58

Age - Specific Means & Standard Deviations of Coronary Risk Factors by Plasma Cholesterol Level

		Plasma Cholesterol < 200 mg%						Plasma Cholesterol 200 - 219 mg%						Plasma Cholesterol ⩾ 220 mg%					
		30 - 39		40 - 49		50 - 60		30 - 39		40 - 49		50 - 60		30 - 39		40 - 49		50 - 60	
		$\bar{x}$	S.D	$\bar{x}$	S.D	$\bar{x}$	S.D	$\bar{x}$	S.D	$\bar{x}$	S.D	$\bar{x}$	S.D	$\bar{x}$	S.D	$\bar{x}$	S.D	$\bar{x}$	S.D
Height	M	171.70	5.98	172.21	5.56	169.52	5.45	172.11	5.95	172.12	5.94	170.71	6.19	172.75	5.38	172.71	5.41	171.02	6.13
	F	161.64	5.45	162.36	4.76	162.54	3.11	162.13	4.92	163.07	4.73	160.11	4.71	162.29	5.95	161.68	6.05	160.84	5.12
Weight	M	71.41	9.57	79.68	12.46	72.03	10.37	78.78	15.87	81.62	11.11	75.32	9.36	85.89	6.97	86.69	10.84	79.02	13.01
	F	64.35	7.98	65.08	8.31	69.12	7.39	71.83	9.21	70.61	9.81	63.87	8.67	78.04	9.31	73.32	12.17	69.22	8.56
BMI (Weight/ Height2)	M	24.21	3.05	27.13	4.77	24.66	3.79	26.64	4.92	27.58	4.11	25.85	4.99	28.89	4.81	29.29	4.47	26.97	4.13
	F	24.62	4.43	24.76	4.68	26.18	4.96	27.58	4.89	26.49	4.11	24.91	3.09	28.16	4.88	28.06	4.56	26.74	3.99
Systolic B.P.	M	123.69	9.09	126.69	11.87	128.66	11.95	127.37	10.75	131.39	13.54	134.05	15.14	134.14	13.61	135.17	17.31	140.21	16.44
	F	118.81	13.24	123.89	7.59	126.07	10.91	122.11	11.09	125.45	9.64	136.15	16.48	122.15	10.45	135.38	15.21	140.19	15.61
Diastolic B.P.	M	79.98	5.28	87.38	7.52	82.93	7.31	82.04	6.19	87.48	5.18	86.42	10.45	85.58	7.76	85.05	8.23	87.42	8.73
	F	76.05	6.76	81.32	7.52	79.28	6.11	78.48	7.65	81.41	5.18	102.05	28.86	78.98	7.65	84.05	8.04	15.91	9.11
Fasting Blood Sugar	M	86.05	8.62	96.57	31.67	94.62	23.38	94.22	11.91	96.75	14.82	96.98	20.51	101.65	16.56	99.52	20.92	115.64	54.26
	F	85.83	10.01	103.88	39.15	88.71	16.87	92.53	8.71	96.35	13.92	101.95	31.03	107.36	26.15	110.26	45.02	107.28	48.07

TABLE 59

DISTRIBUTION OF STUDY POPULATION BY AGE, SEX & BLOOD PRESSURE LEVELS*

		MALE			FEMALE			TOTAL	
		30-39	40-49	50-60	30-39	40-49	50-60	MALE	FEMALE
Normotensive	No	487	311	165	468	216	114	963	798
	%	78.9	64.5	47.8	92.5	79.7	61.3	66.7	82.9
Borderline hypertensive	No	101	119	118	20	36	44	338	100
	%	16.4	24.7	34.2	4.0	13.3	23.7	23.4	10.4
Hypertensive	No	29	52	62	18	19	28	143	65
	%	4.7	10.8	18.0	3.6	7.0	15.1	9.9	6.7
TOTAL	No	617	482	345	506	271	186	1444	963
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

* Subjects grouped according to blood pressure levels based on the WHO classification.
Normotensive: Systolic blood pressure $<$140 mm Hg and diastolic blood pressure $<$90 mm Hg.
Borderline hypertensive: Systolic blood pressure 140-159 mm Hg and diastolic $<$95 mm Hg, or Diastolic 90-94 mm Hg and systolic $<$160 mm Hg.
Hypertensive: Systolic pressure 160 mm Hg or over, or diastolic 95 mm Hg and over.

TABLE 60

Prevalence of Angina & Hypertension

Age & Sex		Normotensive			Borderline Hypertensive			Hypertensive		
		N	ANGINA No	%	N	ANGINA No	%	N	ANGINA No	%
30-39	M	487	10	2.1	101	1	1.0	29	2	6.9
	F	468	2	0.4	20	0	0	18	2	11.1
40-49	M	311	6	1.9	119	5	4.2	52	7	13.5
	F	216	3	1.4	36	1	2.8	19	3	15.8
50-60	M	165	6	3.6	118	6	5.1	62	12	19.4
	F	114	2	1.8	44	1	2.3	28	5	17.9
Total	M	963	22	2.3	338	12	3.6	143	21	14.7
	F	798	7	0.9	100	2	2.0	65	10	15.4
Age-adjusted	M			2.4			3.1			12.1
	F			0.9			1.2			13.7

Significance of hypertension controlling age and sex $P<0.001$

TABLE 60

Prevalence of Angina & Hypertension

Age & Sex		Normotensive			Borderline Hypertensive			Hypertensive		
		N	ANGINA No	%	N	ANGINA No	%	N	ANGINA No	%
30-39	M	487	10	2.1	101	1	1.0	29	2	6.9
	F	468	2	0.4	20	0	0	18	2	11.1
40-49	M	311	6	1.9	119	5	4.2	52	7	13.5
	F	216	3	1.4	36	1	2.8	19	3	15.8
50-60	M	165	6	3.6	118	6	5.1	62	12	19.4
	F	114	2	1.8	44	1	2.3	28	5	17.9
Total	M	963	22	2.3	338	12	3.6	143	21	14.7
	F	798	7	0.9	100	2	2.0	65	10	15.4
Age-adjusted	M			2.4			3.1			12.1
	F			0.9			1.2			13.7

Significance of hypertension controlling age and sex $P<0.001$

TABLE 61

Prevalence of PMI & Hypertension

Age & Sex		Normotensive			Borderline Hypertensive			Hypertensive		
		N	PMI No	PMI %	No	PMI No	PMI %	No	PMI No	PMI %
30-39	M	487	2	0.4	101	4	4.0	29	3	10.3
	F	468	2	0.4	20	0	0	18	0	0
40-49	M	311	2	0.6	119	3	2.5	52	5	9.6
	F	216	1	0.5	36	0	0	19	2	10.5
50-60	M	165	5	3.0	118	7	5.9	62	4	6.5
	F	114	0	0	44	1	2.3	28	1	3.6
Total	M	963	9	0.9	338	14	4.1	143	12	8.4
	F	798	3	0.4	100	1	1.0	65	3	4.6
Age-adjusted	M			1.1			3.9			9.2
	F			0.3			0.4			3.6

Significance of hypertension controlling for age and sex $P<0.001$

TABLE 62

Prevalence of Ischaemic-type ECG & Hypertension

Age & Sex		Normotensive			Borderline Hypertensive			Hypertensive		
		N	ECG		N	ECG		N	ECG	
			No	%		No	%		No	%
30-39	M	487	3	0.6	101	5	5.0	29	3	10.3
	F	468	3	0.6	20	1	5.0	18	1	5.6
40-49	M	311	5	1.6	119	7	5.9	52	12	23.1
	F	216	0	0	36	2	5.6	19	3	15.8
50-60	M	165	5	3.0	118	13	11.0	62	16	25.8
	F	114	0	0	44	5	11.4	28	7	25.0
Total	M	963	13	1.3	338	25	7.4	143	31	21.7
	F	798	3	0.4	100	8	8.0	65	11	16.9
Age-adjusted	M			1.5			6.7			18.3
	F			0.3			6.4			12.2

Significance of hypertension controlling for age and sex $P<0.001$

TABLE 63

Prevalence of ischaemic-type ECG without Symptoms & Hypertension

Age & Sex		Normotensive			Borderline Hypertensive			Hypertensive		
		N	ECG without symptoms No	%	N	ECG without symptoms No	%	N	ECG without symptoms No	%
30-39	M	487	1	0.2	101	0	0	29	2	6.9
	F	468	1	0.2	20	1	5.0	18	0	0
40-49	M	311	3	1.0	119	6	5.0	52	5	9.6
	F	216	0	0	36	1	2.8	19	1	5.3
50-60	M	165	3	1.8	118	5	4.2	62	12	19.4
	F	114	0	0	44	4	9.1	28	3	10.7
Total	M	963	7	0.7	338	11	3.3	143	19	13.3
	F	798	1	0.1	100	6	6.0	65	4	6.2
Age-adjusted	M			0.8			2.7			10.8
	F			0.1			5.2			3.6

Significance of hypertension controlling for age and sex $P<0.001$

TABLE 64

Age - Specific Means & Standard Deviations of Coronary Risk Factors by Blood Pressure Level

		Normotensives						Borderline Hypertensives						Hypertensive					
		30 - 39		40 - 49		50 - 60		30 - 39		40 - 49		50 - 60		30 - 39		40 - 49		50 - 60	
		$\bar{x}$	S.D	$\bar{x}$	S.D	$\bar{x}$	S.D	$\bar{x}$	S.D	$\bar{x}$	S.D	$\bar{x}$	S.D	$\bar{x}$	S.D	$\bar{x}$	S.D	$\bar{x}$	S.D
Height	M	171.99	5.89	172.37	5.46	169.88	5.74	171.76	6.31	173.28	5.81	172.17	5.65	172.38	3.56	170.54	5.59	169.68	6.88
	F	161.78	5.35	162.49	5.19	160.64	4.28	163.81	7.18	160.75	4.99	161.32	5.61	161.61	3.27	162.63	7.21	162.43	4.73
Weight	M	75.63	4.61	76.63	11.83	74.28	10.21	77.27	10.71	86.90	9.77	79.91	7.67	78.68	10.84	92.17	4.25	77.04	12.34
	F	67.81	6.66	69.08	10.21	66.38	7.74	64.55	5.09	72.27	10.92	68.88	8.48	77.86	11.01	78.94	7.71	73.71	9.61
BMI (Weight/ Height^2)	M	25.58	4.09	25.77	3.63	25.22	4.99	26.19	3.29	29.89	4.24	26.92	4.43	25.77	3.32	30.18	4.27	26.73	3.85
	F	24.70	3.27	26.16	3.67	25.71	4.67	26.29	3.75	27.04	5.24	26.45	3.76	26.05	4.91	28.89	4.11	28.01	4.28
Plasma Cholesterol	M	196.34	21.31	210.05	25.78	217.21	27.61	214.09	26.48	223.85	27.31	232.46	39.19	221.37	29.17	227.09	29.87	235.11	29.02
	F	193.63	21.42	215.89	28.07	216.48	21.65	208.05	17.58	221.27	19.61	233.49	26.66	211.82	20.84	240.15	28.31	238.03	22.83
Fasting Blood Glucose	M	90.12	13.12	94.74	16.48	100.09	26.72	94.32	11.29	105.81	35.30	109.52	38.02	94.24	10.23	98.27	17.39	117.83	44.97
	F	89.61	10.14	103.45	60.73	93.73	15.69	113.21	51.93	110.88	50.52	112.02	34.56	97.17	30.47	113.57	22.61	119.71	38.68

TABLE 65

DISTRIBUTION OF PERSONS BY SMOKING CATEGORY, AGE AND SEX

	30 - 39 years				40 - 49 years				50 - 60 years				All ages			
	Male		Female		Male		Female		Male		Female		Male		Female	
	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Smokers	285	46.3	63	12.4	228	47.3	40	14.7	159	46.1	38	20.4	673	46.6	141	14.6
Ex-smokers	82	13.3	10	2.0	67	13.9	19	7.1	59	17.1	7	3.8	208	14.4	36	3.7
Never smoked	249	40.4	433	85.6	187	38.8	212	78.2	127	36.8	141	75.8	563	38.9	786	81.6
Study Population	617	100.0	506	100.0	482	100.0	271	100.0	345	100.0	186	100.0	1444	100.0	963	100.0

TABLE 66

DISTRIBUTION OF CIGARETTES SMOKED BY AGE & SEX

	30-39 years				40-49 years				50-60 years				ALL AGES			
	MALE		FEMALE		MALE		FEMALE		MALE		FEMALE		MALE		FEMALE	
	No	%	No	%	No	%	No	%	No	%	No	%	No	%	No	%
1-4 cigarettes/day	9	3.15	9	14.28	7	3.07	6	15.0	0	0	0	0	16	2.38	15	10.64
5-14 cigarettes/day	39	13.64	16	25.39	19	8.33	8	20.00	10	6.29	1	2.63	68	10.11	25	17.73
15-24 cigarettes/day	135	47.21	26	41.27	118	51.75	24	60.00	87	54.72	31	81.58	340	50.52	81	57.45
25 + cigarettes/day	103	36.01	12	19.05	84	36.84	2	5.00	62	38.99	6	15.79	249	36.99	20	14.18
TOTAL	286	100.00	63	100.00	228	100.00	40	100.00	159	100.00	38	100.00	673	100.00	141	100.00

TABLE 67

PREVALENCE OF ANGINA AND PMI SMOKING

Age and Sex		ANGINA										PMI								
		N	Never smoked		N	Ex-smokers		N	smokers		N	Never smoked		N	Ex-smokers		N	smokers	
			No	%		No	%		No	%		No	%		No	%		No	%
30-39	M	249	4	1.6	82	4	4.9	286	5	1.7	249	4	1.6	82	2	2.4	286	3	1.0
	F	433	2	0.5	10	0	0	63	2	3.2	433	1	0.2	10	0	0	63	1	1.6
40-49	M	187	7	3.7	67	3	4.5	228	8	3.5	187	2	1.1	67	4	6.0	228	4	1.8
	F	212	3	1.4	19	2	10.5	40	2	5.0	212	1	0.5	19	0	0	40	2	5.0
50-60	M	127	3	2.4	59	7	11.9	159	14	8.8	127	1	0.8	59	9	15.3	159	6	3.8
	F	141	2	1.4	7	0	0	38	6	15.8	141	2	1.4	7	0	0	38	0	0
Total	M	563	14	2.5	208	14	6.7	673	27	4.0	563	7	1.2	208	15	7.2	673	13	1.9
	F	786	7	0.9	36	2	5.6	141	10	7.1	786	4	0.5	36	0	0	141	3	2.1
Age-adjusted	M			2.5			6.4			4.0			1.2			6.7			1.9
	F			0.9			2.9			6.1			0.5			0.0			2.2

Significance of smoking controlling for age and sex: ANGINA $P<0.001$; PMI $P<0.01$

TABLE 68

Prevalence of ischemiac-type ECG changes and ischaemic-type ECG changes without symptoms and smoking

Age and Sex		Ischaemic-type ECG changes									Ischaemic-type ECG changes without symptoms								
		N	Never smoked		N	Ex-smokers		N	smokers		N	Never smoked		N	Ex-smokers		N	smokers	
			No	%		No	%		No	%		No	%		No	%		No	%
30-39	M	249	4	1.6	83	2	2.4	286	5	1.7	249	4	1.6	82	0	0	286	3	1.0
	F	433	2	0.5	10	0	0	63	2	3.2	433	1	0.2	10	0	0	63	1	1.6
40-49	M	187	7	3.7	67	8	11.9	228	3	3.5	187	2	1.1	67	5	7.5	228	4	1.8
	F	212	3	1.4	19	0	0	40	2	5.0	212	1	0.5	19	0	0	40	2	5.0
50-60	M	127	3	2.4	59	13	22.0	159	14	8.8	127	1	0.8	59	5	8.5	159	6	3.8
	F	141	2	1.4	7	1	14.3	38	6	15.8	141	2	1.4	7	1	14.3	38	0	0
Total	M	563	14	2.5	208	23	11.1	673	27	4.0	563	7	1.2	208	10	4.8	673	13	1.9
	F	786	7	0.9	35	1	2.8	141	10	7.1	786	4	0.5	35	1	2.8	141	3	2.1
Age-adjusted	M			3.3			10.2			4.0			2.1			4.5			2.2
	F			1.1			2.7			7.7			0.5			2.8			3.6

Significance of smoking controlling for age and sex $P < 0.001$ $P < 0.05$

TABLE 69

Age-Specific Means and Standard Deviations of Coronary Risk Factors by Smoking Habits and Sex

		SMOKERS						EX-SMOKERS						NEVER SMOKED					
		30 - 39		40 - 49		50 - 60		30 - 39		40 - 49		50 - 60		30 - 39		40 - 49		50 - 60	
		$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.	$\bar{X}$	S.D.
Height (cms)	M	172.39	5.90	172.61	5.66	170.71	5.71	171.87	5.91	172.49	5.59	170.62	6.20	171.61	6.42	172.20	5.54	170.55	6.28
	F	163.47	5.61	162.67	5.89	161.65	5.74	165.31	4.74	163.68	8.31	159.71	7.76	161.62	5.37	162.21	5.24	160.92	4.42
Weight (kgms)	M	74.41	9.49	78.98	7.58	76.21	14.62	78.51	9.35	79.99	11.90	77.32	10.36	78.93	10.22	86.91	7.41	77.13	10.38
	F	64.51	10.36	70.10	12.46	67.61	7.01	63.41	7.85	74.11	13.54	70.28	15.94	68.73	8.79	70.23	12.55	68.19	8.95
BMI (Weight/ height^2)	M	26.44	3.08	27.67	4.38	26.10	4.45	26.22	4.95	28.29	4.03	27.52	4.07	26.04	3.05	28.49	4.53	26.47	4.98
	F	24.11	3.56	26.42	3.21	25.85	3.14	23.21	3.78	27.79	4.85	27.31	4.19	26.46	3.86	26.71	4.73	26.33	3.27
Systolic B.P.	M	122.41	9.49	128.98	14.46	138.41	16.09	127.19	11.44	133.28	17.22	144.66	16.88	126.71	15.49	131.55	16.49	134.06	15.91
	F	117.51	10.36	123.10	12.46	133.15	16.98	118.51	14.72	129.21	16.44	131.43	14.64	119.61	11.83	128.37	12.04	135.64	15.58
Diastolic B.P.	M	81.41	6.32	89.67	7.98	87.31	8.81	83.04	6.88	84.02	8.45	87.71	10.84	81.63	6.43	83.52	7.88	85.01	9.11
	F	78.33	9.95	84.25	9.57	84.73	10.19	78.12	11.35	80.26	7.35	81.42	6.91	76.87	6.73	82.21	6.35	88.58	6.40
Plasma cholesterol	M	202.34	23.89	219.54	26.75	232.35	31.36	202.31	26.21	219.08	27.37	226.25	29.22	198.77	23.97	212.38	27.92	225.03	34.74
	F	200.47	19.83	226.92	21.11	230.55	22.27	223.41	25.19	230.11	32.54	251.85	32.27	193.41	21.51	216.82	28.52	221.14	25.30
Fasting blood glucose	M	91.39	12.99	96.71	19.41	103.40	33.66	93.14	16.97	94.59	15.87	110.27	36.83	97.66	12.67	98.88	52.34	109.16	72.81
	F	90.20	9.57	96.97	8.56	105.71	29.24	89.41	5.96	112.11	51.98	119.71	17.32	90.89	16.53	105.74	62.16	101.01	41.80

TABLE 70

SMOKING HABITS OF PATIENTS WITH INTERMITTENT CLAUDICATION (IC)

	No. of patients with IC	
	Males	Females
Never smoked	1	0
Ex-smokers	3	2
Current smokers	16	4
T O T A L	20	6

TABLE 71

ACTIVITIES OF VIGOROUS EXERCISE

Activity	Examples of "Vigorous Exercise"
Recreation	Swimming, tennis, sailing as crew; hill-climbing; dancing (specified)
"Keep-fit"	Morning exercises, 5BX*
Physical work ≠	
Gardening	Planting bushes, clearing scrub, felling trees; any work on own allotment
Do-it-yourself	Building in stone or concrete/ demolition
Other	Moving heavy objects; major, rusted, car repairs
Getting about	Brisk walking in town, over rough country; running; cycling (not otherwise specified) ≠
Climbing up stairs	500+ daily

* Royal Canadian Air Force fitness system

≠ Spell of over 30 minutes, and/or total of 1 hour or more.

TABLE 72

EXERCISE IN LEISURE-TIME BY AGE AND SEX

		30 - 39 years		40 - 49 years		50 - 60 years		T O T A L	
		Males	Females	Males	Females	Males	Females	Males	Females
Doing Vigorous Exercise	No.	313	238	132	73	73	45	518	356
	%	50.7	47.0	27.4	26.9	21.2	24.2	35.9	37.0
Not Doing Vigorous Exercise	No.	304	268	350	198	272	141	926	607
	%	49.3	53.0	72.6	73.1	78.8	75.8	64.1	63.0
TOTAL	No.	617	506	482	271	345	186	1444	963
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 73

FREQUENCY OF EXERCISE IN LEISURE-TIME BY AGE AND SEX

		30 - 39 years		40 - 49 years		50 - 60 years		T O T A L	
		Males	Females	Males	Females	Males	Females	Males	Females
Daily	No.	69	51	36	14	22	9	127	74
	%	22.1	21.4	27.3	19.2	30.1	20.0	24.5	20.8
Weekly	No.	63	33	35	3	23	16	121	52
	%	20.1	13.9	26.5	4.1	31.5	35.6	23.4	14.6
Occasionally	No.	181	154	61	56	28	20	270	230
	%	57.8	64.7	46.2	76.7	38.4	44.4	52.1	64.6
T O T A L	No.	313	238	132	73	73	45	518	356
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 74

Prevalence of Angina and PMI and Exercise in leisure time

Age and Sex		ANGINA						PMI					
		N	Doing exercise No.	%	N	Not doing exercise No.	%	N	Doing exercise No.	%	N	Not doing exercise No.	%
30 - 39	M	313	3	0.9	304	10	3.3	313	2	0.6	304	7	2.3
	F	238	1	0.4	268	3	1.1	238	0	0	268	2	0.7
40 - 49	M	132	0	0	350	18	5.1	132	0	0	350	10	2.8
	F	73	0	0	198	7	3.5	73	0	0	198	3	1.5
50 - 60	M	73	0	0	272	24	8.8	73	0	0	272	16	5.9
	F	45	0	0	141	8	5.6	45	0	0	141	2	1.4
Total	M	518	3	0.6	926	52	5.6	518	2	0.4	926	33	3.5
	F	356	1	0.6	307	18	2.9	356	0	0	607	7	1.1
Age-adjusted	M			0.4			5.2			0.3			3.3
	F			0.2			2.6			0.0			1.1
Significance of exercise controlling for age and sex				P<0.001						P<0.001			

TABLE 75

Prevalence of ischaemic-type ECG changes and ischaemic-type ECG changes without symptoms and exercise in leisure time

Age and Sex		Ischaemic-type ECG changes						Ischaemic-type ECG changes without symptoms					
		Doing exercise			Not doing exercise			Doing exercise			Not doing exercise		
		N	No.	%	N	No.	%	N	No.	%	N	No.	%
30 - 39	M	313	0	0	304	11	3.6	313	0	0	304	3	0.9
	F	238	0	0	268	5	1.8	238	0	0	268	2	0.7
40 - 49	M	132	0	0	350	24	6.8	132	0	0	350	14	4.0
	F	73	1	1.4	198	4	2.0	73	1	1.4	198	1	0.5
50 - 60	M	73	2	2.7	272	32	11.7	73	2	2.7	272	18	6.6
	F	45	0	0	141	12	8.5	45	0	0	141	7	4.9
Total	M	518	2	0.4	926	67	7.2	518	2	0.4	926	35	3.8
	F	356	1	0.3	607	21	3.4	356	1	0.3	607	10	1.6
Age-adjusted	M			0.6			6.6			0.6			3.3
	F			0.4			3.1			0.4			1.4
Significance of exercise controlling for age and sex				P<0.001						P<0.001			

TABLE 76

Age-Specific Means & Standard Deviations of
Coronary Risk Factors by Vigorous Exercise & Sex

		Doing Exercise						Not Doing Exercise					
		30-39		40-49		50-60		30-39		40-49		50-60	
		$\bar{x}$	S.D	$\bar{x}$	S.D	$\bar{x}$	S.D	$\bar{x}$	S.D	$\bar{x}$	S.D	$\bar{x}$	S.D
Height (cms)	M	172.06	6.01	172.73	5.16	170.74	4.77	171.87	5.75	172.26	5.76	170.59	6.31
	F	162.28	5.34	161.52	4.71	159.56	3.95	161.47	5.49	162.56	5.53	161.55	4.84
Weight (KGMs)	M	74.53	10.91	76.52	10.42	74.62	11.26	77.41	10.56	85.66	7.36	77.27	12.78
	F	67.21	7.42	68.22	10.05	66.27	8.41	68.31	7.74	71.29	13.16	68.65	8.58
BMI (Weight/ Height^2)	M	25.18	4.44	25.61	4.96	25.54	4.28	26.22	4.33	29.12	4.31	26.51	3.82
	F	25.99	3.52	25.77	3.74	26.01	3.88	26.32	3.82	27.01	4.91	26.31	4.15
Systolic B.P.	M	125.72	10.38	129.39	12.91	134.66	12.51	127.38	11.86	132.36	15.92	136.84	16.76
	F	121.02	13.14	127.46	8.42	132.33	7.81	119.22	11.79	129.92	14.32	137.38	17.52
Diastolic B.P.	M	81.12	6.06	84.65	5.02	85.04	6.65	81.96	6.68	86.73	9.11	86.82	9.46
	F	77.99	7.19	80.96	5.44	79.89	5.06	76.23	7.15	83.09	7.34	90.32	8.02
Plasma cholesterol	M	197.88	21.47	210.06	27.61	215.26	26.41	203.05	26.11	217.27	27.46	228.43	34.29
	F	193.62	21.55	213.05	21.58	218.56	24.89	194.89	21.32	220.25	29.52	224.51	24.88
Fasting Blood Glucose	M	90.12	10.72	94.68	13.78	94.91	13.73	91.91	14.64	99.05	46.44	109.81	64.72
	F	90.09	10.23	95.29	14.06	90.06	6.79	91.44	19.49	107.83	66.51	105.77	44.62

TABLE 77

Age-adjusted Percents of Coronary Risk Factors by Vigorous Exercise & Sex

	MALES		FEMALES	
	Doing Exercise	Not doing Exercise	Doing Exercise	Not doing Exercise
Height Percent $\geq$ 176 cms (Males) Percent $\geq$ 166 cms (Females)	20.9	21.3	15.8	18.6
BMI (Weight/Height^2) Percent $\geq$ 28	15.1	25.5	14.1	20.4
Blood Pressure Percent Normotensive	73.4	63.6	90.6	81.8
Percent Borderline Hypertensive	20.3	25.2	6.8	10.3
Percent Hypertensive	6.3	11.2	2.6	7.9
Plasma cholesterol Percent $\geq$ 220 mg%	29.5	37.7	28.7	32.7
Fasting Blood Glucose Percent $\geq$ 120 mg%	2.1	5.4	0.4	6.0
Percent smokers	40.6	49.4	12.6	15.8
Percent ex smokers	16.2	13.1	2.3	3.9
Behaviour Pattern Percent Type A	53.8	38.3	57.5	35.8
Bread				
White	43.4	49.9	49.3	51.8
Brown	14.9	11.2	13.2	9.4
Both	41.6	38.9	38.5	38.8

TABLE 78

BEING BOSSY OR DOMINATING

		30 - 39		40 - 49		50 - 60		N	
		Male	Female	Male	Female	Male	Female	Male	Female
Very well	No.	69	20	84	42	63	19	216	81
	%	11.2	4.0	17.4	15.5	18.3	10.2	15.0	8.4
Fairly well	No.	233	192	187	122	142	88	562	402
	%.	37.8	37.9	38.8	45.0	41.2	47.3	38.9	41.7
Somewhat	No.	143	142	129	66	76	53	348	261
	%	23.2	28.1	26.8	24.4	22.0	28.5	24.1	27.1
Not at all	No.	172	152	82	41	64	26	318	219
	%	27.9	30.0	17.0	15.1	18.6	14.0	22.0	22.7
Total	No.	617	506	482	271	345	186	1444	963
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 78

BEING BOSSY OR DOMINATING

		30 - 39		40 - 49		50 - 60		N	
		Male	Female	Male	Female	Male	Female	Male	Female
Very well	No.	69	20	84	42	63	19	216	81
	%	11.2	4.0	17.4	15.5	18.3	10.2	15.0	8.4
Fairly well	No.	233	192	187	122	142	88	562	402
	%.	37.8	37.9	38.8	45.0	41.2	47.3	38.9	41.7
Somewhat	No.	143	142	129	66	76	53	348	261
	%	23.2	28.1	26.8	24.4	22.0	28.5	24.1	27.1
Not at all	No.	172	152	82	41	64	26	318	219
	%	27.9	30.0	17.0	15.1	18.6	14.0	22.0	22.7
Total	No.	617	506	482	271	345	186	1444	963
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 79

HAVING A STRONG NEED TO EXCEL IN MOST THINGS

		30 - 39		40 - 49		50 - 60		N	
		Male	Female	Male	Female	Male	Female	Male	Female
Very well	No.	395	280	211	93	127	44	733	417
	%	64.0	55.3	43.8	34.3	36.8	23.7	50.8	43.3
Fairly well	No.	145	160	170	100	121	87	436	347
	%	23.5	31.6	35.3	36.9	35.1	46.8	30.2	36.0
Somewhat	No.	47	49	73	60	64	41	184	150
	%	7.6	9.7	15.1	22.1	18.6	22.0	12.7	15.6
Not at all	No.	30	17	28	18	33	14	91	49
	%	4.9	3.4	5.8	6.6	9.6	7.5	6.3	5.1
Total	No.	617	506	482	271	345	186	1444	963
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 80

USUALLY FEELING PRESSED FOR TIME

		30 - 39		40 - 49		50 - 60		N	
		Male	Female	Male	Female	Male	Female	Male	Female
Very well	No.	126	141	92	48	50	23	268	212
	%	20.4	27.7	19.1	17.7	14.5	12.4	18.6	21.9
Fairly well	No.	187	151	156	94	94	58	437	303
	%	30.3	29.8	32.4	34.7	27.2	31.2	30.3	31.5
Somewhat	No.	158	149	134	89	120	75	412	313
	%	25.6	29.4	27.8	32.8	34.8	40.3	28.5	32.5
Not at all	No.	146	65	100	40	81	30	327	135
	%	23.7	12.8	20.7	14.8	23.5	16.1	22.6	14.0
Total	No.	617	506	482	271	345	186	1444	963
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 81

BEING HARD DRIVING AND COMPETITIVE

		30 - 39		40 - 49		50 - 60		N	
		Male	Female	Male	Female	Male	Female	Male	Female
Very well	No.	374	240	189	64	107	31	670	335
	%	60.6	47.4	39.2	23.6	31.0	16.7	46.4	34.8
Fairly well	No.	162	187	153	127	107	78	422	392
	%	26.3	37.0	31.7	46.9	31.0	41.9	29.2	40.7
Somewhat	No.	47	53	93	59	72	48	212	160
	%	7.6	10.5	19.3	21.8	20.9	25.8	14.7	16.6
Not at all	No.	34	26	47	21	59	29	140	76
	%	5.5	5.1	9.8	7.7	17.1	15.6	9.7	7.9
Total	No.	617	506	482	271	345	186	1444	963
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 82

EATING TOO QUICKLY

		30 - 39		40 - 49		50 - 60		N	
		Male	Female	Male	Female	Male	Female	Male	Female
Very well	No.	153	111	86	27	59	6	298	144
	%	24.8	21.9	17.8	10.0	17.1	3.2	20.6	15.0
Fairly well	No.	236	207	193	83	94	54	523	344
	%	38.2	40.9	40.0	30.6	27.2	29.0	36.2	35.7
Somewhat	No.	120	100	138	97	123	80	381	277
	%	19.4	19.8	28.6	35.8	35.7	43.0	26.4	28.8
Not at all	No.	108	88	65	64	69	46	242	198
	%	17.5	17.4	13.5	23.6	20.0	24.7	16.8	20.6
Total	No.	617	506	482	271	345	186	1444	963
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 83

OFTEN FELT VERY PRESSED FOR TIME

		30 - 39		40 - 49		50 - 60		N	
		Male	Female	Male	Female	Male	Female	Male	Female
Yes	No.	191	199	151	100	116	71	458	370
	%	31.0	39.3	31.3	36.9	33.6	38.2	31.7	38.4
No	No.	426	307	331	171	229	115	986	593
	%	69.0	60.7	68.7	63.1	66.4	61.8	68.3	61.6
Total	No.	617	506	482	271	345	186	1444	963
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 84

OFTEN THINKING ABOUT WORK AFTER WORKING HOURS

		30 - 39		40 - 49		50 - 60		N	
		Male	Female	Male	Female	Male	Female	Male	Female
Yes	No.	285	241	169	92	129	75	583	408
	%	46.2	47.6	35.1	33.9	37.4	40.3	40.4	42.4
No	No.	332	265	313	179	216	111	861	555
	%	53.8	52.4	64.9	66.1	62.6	59.7	59.6	57.6
Total	No.	617	506	482	271	345	186	1444	963
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 85

OFTEN STRETCHED BY WORK TO THE VERY LIMITS OF ENERGY AND CAPACITY

		30-39		40-49		50-60		N	
		Male	Female	Male	Female	Male	Female	Male	Female
Yes	No.	333	289	200	98	141	49	674	429
	%	54.0	55.7	41.5	36.2	40.9	26.3	46.7	44.5
No	No.	284	224	282	173	204	137	770	534
	%	46.0	44.3	58.5	63.8	59.1	73.7	53.3	55.5
Total	No.	617	506	482	271	345	186	1444	963
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 86

OFTEN FELT UNCERTAIN OR DISSATISFIED ABOUT PERFORMANCE OF WORK

		30 - 39		40 - 49		50 - 60		N	
		Male	Female	Male	Female	Male	Female	Male	Female
Yes	No.	171	123	121	59	78	43	370	225
	%	27.7	24.3	25.1	21.8	22.6	23.1	25.6	23.4
No	No.	446	383	361	212	267	143	1074	738
	%	72.3	75.7	74.9	78.2	77.4	76.9	74.4	76.6
Total	No.	617	506	482	271	345	186	1444	963
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 87

GETTING QUITE UPSET WHEN WAITING FOR ANYTHING

		30 - 39		40 - 49		50 - 60		N	
		Male	Female	Male	Female	Male	Female	Male	Female
Yes	No.	413	378	303	186	217	118	933	682
	%	66.9	74.7	62.9	68.6	62.9	63.4	64.6	70.8
No	No.	204	128	179	85	128	68	511	281
	%	33.1	25.3	37.1	31.4	37.1	36.6	35.4	29.2
Total	No.	617	506	482	271	345	186	1444	963
	%	100.0	100.0	100.0	100.0	100.0	100.0	100.0	100.0

TABLE 88

Scoring Scale for Framingham Type A Behavior Pattern Questions

	Very well	Fairly well	Some-what	Not at all
1. Being bossy or dominating	1	.67	.33	0
2. Having a strong need to excel (be best) in most things	1	.67	.33	0
3. Usually feeling pressed for time	1	.67	.33	0
4. Being hard driving and competitive	1	.67	.33	0
5. Eating too quickly	1	.67	.33	0

	Yes	No
6. Have you often felt pressed for time?	1	0
7. Has your work often stayed with you so that you were thinking about it after working hours?	1	0
8. Has your work often stretched you to the very limits of your energy and capacity?	1	0
9. Have you often felt uncertain, uncomfortable or dissatisfied with how well you were doing in your regular line of work?	1	0
10. Do you get quite upset when you have to wait for anything?	1	0

N.B. Because of the variety of item responses throughout the questionnaire i.e. yes-no or multiple choice response sets, scales were scored by summing the responses to each question (valued from 0 to 1, meaning complete presence of a trait) and dividing by the number of questions.

TABLE 89

Prevalence of Angina and PMI and Type of Behaviour

Age and Sex		ANGINA						PMI					
		N	Type B No.	%	N	Type A No.	%	N	Type B No.	%	N	Type A No.	%
30 - 39	M	270	3	1.1	347	10	2.9	270	4	1.5	347	5	1.4
	F	217	1	0.5	289	3	1.0	217	0	0	289	2	0.7
40 - 49	M	247	8	3.2	235	10	4.3	247	2	0.8	235	8	3.4
	F	144	4	2.8	127	3	2.4	144	0	0	127	3	2.4
50 - 60	M	205	10	4.9	140	14	10.0	205	4	2.0	140	12	8.6
	F	101	1	1.0	85	7	8.2	101	0	0	85	2	2.4
Total	M	722	21	2.9	722	34	4.7	722	10	1.4	722	25	3.5
	F	462	6	1.3	501	13	2.6	462	0	0	501	7	1.4
Age-adjusted	M			2.7			5.1			1.4			3.8
	F			1.2			2.8			0.0			1.5

Significance of behaviour pattern controlling for age and sex $0.1 > P > 0.05$ $P < 0.01$

TABLE 90

Prevalence of ischaemic-type ECG changes and ischaemic-type ECG changes without symptoms and type of behaviour

Age and Sex		Ischaemic-type ECG changes						Ischaemic-type ECG changes without symptoms					
		N	Type B No.	%	N	Type A No.	%	N	Type B No.	%	N	Type A No.	%
30 - 39	M	270	6	2.2	347	5	1.4	270	2	0.7	347	1	0.3
	F	217	1	0.5	289	4	1.4	217	1	0.5	289	1	0.3
40 - 49	M	247	6	2.4	235	18	7.7	247	3	1.2	235	11	4.7
	F	144	0	0	127	5	3.9	144	0	0	127	2	1.5
50 - 60	M	205	17	8.3	140	17	12.1	205	11	5.4	140	9	6.4
	F	101	1	1.0	85	11	12.9	101	1	1.0	85	6	7.1
Total	M	722	29	4.0	722	40	5.5	722	10	2.2	722	21	2.9
	F	462	2	0,4	501	20	4.0	462	2	0.4	501	9	1.8
Age-adjusted	M			3,7			6.1			2.2			3.2
	F			0.4			4.3			0.4			1.9
Significance of behaviour pattern controlling for age and sex				P <0.01						>0.1			

TABLE 91

Prevalence of IHD & type of behaviour - Males

AGE & TYPE OF BEHAVIOUR		N	ANGINA		PMI		ECG		ECG without symptoms	
			No	%	No	%	No	%	No	%
30-38	Type A	347	10	2.9	5	1.4	5	1.4	1	0.3
	Type B	270	3	1.1	4	1.5	6	2.2	2	0.7
40-49	Type A	235	10	4.3	8	3.4	18	7.7	11	4.7
	Type B	247	8	3.2	2	0.8	6	2.4	3	1.2
50-60	Type A	140	14	10.0	12	8.6	17	12.1	9	6.4
	Type B	205	10	4.9	4	2.0	17	8.3	11	5.4
Total	Type A	722	34	4.7	25	3.5	40	5.5	21	2.9
	Type B	722	21	2.9	10	1.4	29	4.0	10	2.2
Age-adjusted	Type A			5.1		3.8		6.1		3.2
	Type B			2.7		1.4		3.7		2.2
Significance of behaviour pattern controlling for age			P<0.05		P<0.01		P<0.05		P>0.1	

TABLE 92

Prevalence of IHD & type of behaviour - Females

AGE & TYPE OF BEHAVIOUR		N	ANGINA		PMI		ECG		ECG without symptoms	
			No	%	No	%	No	%	No	%
30-39	Type A	289	3	1.0	2	0.7	4	1.4	1	0.3
	Type B	217	1	0.5	0	0	1	0.5	1	0.5
40-49	Type A	127	3	2.4	3	2.4	5	3.9	2	1.5
	Type B	144	4	2.8	0	0	0	0	0	0
50-60	Type A	85	7	8.2	2	2.4	11	12.9	6	7.1
	Type B	101	1	1.0	0	0	1	1.0	1	1.0
Total	Type A	501	13	2.6	7	1.4	20	4.0	9	1.8
	Type B	462	6	1.3	0	0	2	0.4	2	0.4
Age-adjusted	Type A			2.8		1.5		4.3		1.9
	Type B			1.2		0.0		0.4		0.4
Significance of behaviour pattern controlling for age				1>P>0.05		P<0.01		P<0.001		P<0.05

TABLE 93

AGE ADJUSTED PERCENTS OF CORONARY RISK FACTORS
BY BEHAVIOUR PATTERN AND SEX

	Males		Females	
	Type A	Type B	Type A	Type B
BMI (weight/height^2) Percent ≥28	22.8	21.1	17.5	19.1
Blood pressure Percent normotensive	65.6	68.6	79.2	86.6
Percent borderline hypertensive	23.5	22.6	12.1	8.9
Percent hypertensive	10.8	8.8	8.7	4.4
Plasma cholesterol Percent ≥220mg%	42.1	28.3	36.6	27.3
Fasting blood glucose Percent ≥120mg%	6.1	3.1	3.9	4.9
Smoking Percent smokers	48.1	45.1	19.9	9.1
percent Ex-smokers	16.5	11.7	3.9	3.4
percent never smoked	35.4	43.2	76.2	87.5
Exercise in leisure time percent doing exercise	38.9	34.1	39.2	34.5

TABLE 93

AGE ADJUSTED PERCENTS OF CORONARY RISK FACTORS
BY BEHAVIOUR PATTERN AND SEX

	Males		Females	
	Type A	Type B	Type A	Type B
BMI (weight/height^2) Percent ≥28	22.8	21.1	17.5	19.1
Blood pressure Percent normotensive	65.6	68.6	79.2	86.6
Percent borderline hypertensive	23.5	22.6	12.1	8.9
Percent hypertensive	10.8	8.8	8.7	4.4
Plasma cholesterol Percent ≥220mg%	42.1	28.3	36.6	27.3
Fasting blood glucose Percent ≥120mg%	6.1	3.1	3.9	4.9
Smoking Percent smokers	48.1	45.1	19.9	9.1
percent Ex-smokers	16.5	11.7	3.9	3.4
percent never smoked	35.4	43.2	76.2	87.5
Exercise in leisure time percent doing exercise	38.9	34.1	39.2	34.5

TABLE 94

Age-specific means & standard deviations of coronary risk factors by behaviour pattern & sex

		MALES						FEMALES					
		30-39		40-49		50-60		30-39		40-49		50-60	
		$\bar{x}$	S.D.	$\bar{x}$	S.D.	$\bar{x}$	S.D.	$\bar{x}$	S.D.	$\bar{x}$	S.D.	$\bar{x}$	S.D.
Height (cms)	Type A	172.42	5.57	172.86	5.25	172.09	5.59	161.97	5.39	162.53	5.46	161.57	5.07
	Type B	171.38	6.19	171.95	5.89	169.62	6.11	161.69	5.48	162.05	5.23	160.64	4.36
Weight (kgms)	Type A	76.49	9.89	85.53	6.62	78.13	10.76	71.23	8.43	69.43	9.75	68.92	9.79
	Type B	74.75	7.32	80.91	9.97	75.73	8.51	64.19	9.15	70.87	8.52	67.36	7.37
BMI (weight/ height^2)	Type A	26.12	2.97	28.83	3.85	26.37	3.32	24.35	3.91	26.31	3.64	26.38	3.52
	Type B	25.43	2.48	27.52	3.52	26.26	4.02	25.59	3.05	26.99	4.38	26.11	2.66
Systolic B.P. (mm Hg)	Type A	126.98	12.04	133.51	16.43	136.71	15.66	120.58	13.77	129.68	11.11	141.60	16.11
	Type B	125.96	9.91	129.67	13.69	135.89	16.45	119.38	10.47	128.89	14.55	131.58	14.17
Diastolic B.P. (mm Hg)	Type A	81.67	6.41	86.04	9.39	86.43	9.47	77.61	7.84	82.61	6.87	86.29	9.39
	Type B	81.35	6.34	86.81	8.19	86.09	8.69	76.31	6.22	82.43	7.02	89.06	8.29
Plasma cholesterol (mg%)	Type A	202.11	24.61	221.58	28.07	231.27	28.89	193.89	21.91	219.22	25.33	232.03	23.07
	Type B	197.91	22.95	209.32	25.64	221.81	29.79	194.83	20.78	217.15	29.79	215.51	24.04
Fasting Blood Glucose	Type A	91.67	12.31	98.08	52.69	106.39	32.15	90.24	17.52	103.22	47.14	107.47	52.77
	Type B	90.15	13.42	97.62	20.27	106.59	40.67	91.56	13.24	105.84	37.51	97.35	22.49

TABLE 95

NATURE OF OCCUPATION, BY AGE AND SEX OF THOSE EXAMINED

	30-39		40-49		50-60		Total	
	No.	%	No.	%	No.	%	No.	%
Males								
(1) Administrative Staff	163	26.4	164	34.1	138	40.0	465	32.2
(2) Teaching Staff	309	50.1	135	28.0	79	22.9	523	36.2
(3) Manual Workers	145	23.5	183	37.9	128	37.1	456	31.6
Total	617	100.0	482	100.0	345	100.0	1444	100.0
Females								
(1) Administrative Staff	147	29.1	80	29.6	56	30.1	282	29.4
(2) Teaching Staff	239	47.2	105	38.7	64	34.4	408	42.4
(3) Manual Workers	120	23.7	86	31.7	66	35.5	272	28.2
Total	506	100.0	271	100.0	186	100.0	963	100.0

TABLE 96

Prevalence of Angina & Occupation

Age & Sex		Administrative Staff			Teaching Staff			Manual Workers		
		N	ANGINA No	%	N	ANGINA No	%	N	ANGINA No	%
30-39	M	163	5	3.1	309	7	2.3	145	1	0.7
	F	147	2	1.4	239	2	0.8	120	0	0
40-49	M	164	7	4.3	135	4	3.0	183	7	3.8
	F	80	2	2.5	105	2	1.9	86	3	3.5
50-60	M	138	12	8.7	79	1	1.3	128	11	8.6
	F	56	5	8.9	64	0	0	66	3	4.5
Total	M	465	24	5.2	523	12	2.3	456	19	4.2
	F	283	9	3.2	408	4	0.9	272	6	2.2
Age-adjusted	M			4.8			2.3			3.6
	F			3.2			0.9			1.8

Significance of occupation controlling for age and sex $P < 0.05$

TABLE 97

Prevalence of Possible Myocardial Infarction & Occupation

Age & Sex		Administrative Staff			Teaching Staff			Manual Workers		
		N	PMI No	PMI %	N	PMI No	PMI %	N	PMI No	PMI %
30-39	M	163	4	2.5	309	4	1.3	145	1	0.7
	F	147	2	1.4	239	0	0	120	0	0
40-49	M	164	7	4.3	135	2	1.5	183	1	0.5
	F	80	2	2.5	105	0	0	86	1	1.2
50-60	M	138	11	8.0	79	0	0	128	5	3.9
	F	56	0	0	64	0	0	66	2	3.0
Total	M	465	22	4.7	523	6	1.1	456	7	1.5
	F	283	4	1.4	408	0	0	272	3	1.1
Age-adjusted	M			4.4			1.1			1.4
	F			1.4			0.00			0.9

Significance of occupation controlling for age and sex $P<0.001$

TABLE 98

Prevalence of ischaemic type ECG & Occupation

Age & Sex		Administrative Staff			Teaching Staff			Manual Workers		
		N	ECG No	ECG %	N	ECG No	ECG %	N	ECG No	ECG %
30-39	M	163	6	3.7	309	4	1.3	145	1	0.7
	F	147	3	2.0	239	2	0.8	120	0	0
40-49	M	164	14	8.5	135	7	5.2	183	3	1.6
	F	80	3	3.7	105	2	1.9	86	0	0
50-60	M	138	23	16.7	79	0	0	128	11	8.6
	F	56	3	5.4	64	4	6.3	66	5	7.6
Total	M	465	43	9.2	523	11	2.1	456	15	3.3
	F	283	9	3.2	408	8	1.9	272	5	1.8
Age-adjusted	M			2.4			2.3			2.9
	F			3.1			2.1			1.5

Significance of occupation controlling for age and sex $P<0.001$

TABLE 99

Prevalence of ischaemic type ECG without symptoms & occupation

Age & Sex		Administrative Staff			Teaching Staff			Manual Workers		
		N	ECG without symptoms No	%	N	ECG without symptoms No	%	N	ECG without symptoms No	%
30-39	M	163	1	0.6	309	2	0.6	145	0	0
	F	147	0	0	239	2	0.8	120	0	0
40-49	M	164	9	5.5	135	3	2.2	183	2	1.1
	F	80	0	0	105	2	1.9	86	0	0
50-60	M	138	12	8.7	79	0	0	128	8	6.3
	F	56	1	1.8	64	4	6.3	66	2	3.0
Total	M	465	22	4.7	523	5	0.9	456	10	2.2
	F	283	1	0.4	408	8	1.9	272	2	0.7
Age-adjusted	M			4.2			0.9			1.9
	F			0.3			2.2			0.6

Significance of occupation controlling for age and sex $P > 0.1$

TABLE 100

Age-adjusted Means, Standard Errors of means and percentages of coronary risk factors by sex and occupation

Variable	Males			Females		
	Administrative	Teaching	Manual	Administrative	Teaching	Manual
Height						
Mean ± SEM	172.6 ± 0.26	171.5 ± 0.29	170.8 ± 0.27	161.8 ± 0.29	161.7 ± 0.24	161.7 ± 0.32
Percent ≥ 176 cms (males)	29.9	22.1	20.5	15.9	17.5	21.9
Percent ≥ 166 cms (females)						
BMI (Weight/Height^2)						
Mean ± SEM	27.3 ± 0.17	25.2 ± 0.82	26.6 ± 0.73	26.0 ± 1.44	25.4 ± 0.76	27.1 ± 1.16
Percent ≥ 28	25.8	17.2	22.8	20.3	11.2	25.8
Systolic B.P.						
Mean ± SEM	130.2 ± 0.65	127.2 ± 0.61	127.7 ± 0.59	126.2 ± 0.88	122.7 ± 0.61	122.4 ± 0.80
Percent ≥ 160	5.8	5.1	6.0	5.9	2.6	3.2
Diastolic B.P.						
Mean ± SEM	81.5 ± 0.34	80.6 ± 0.35	81.9 ± 0.39	78.1 ± 0.48	77.8 ± 0.36	76.6 ± 0.47
Percent ≥ 95	4.9	3.6	6.1	4.2	2.3	2.1
Plasma Cholesterol						
Mean ± SEM	217.5 ± 1.24	211.6 ± 1.18	208.1 ± 1.45	210.3 ± 1.23	104.1 ± 1.13	209.7 ± 1.65
Percent ≥ 220 mg %	42.2	34.8	29.3	38.5	26.9	30.8
Fasting blood glucose						
Mean ± SEM	83.9 ± 0.93	79.2 ± 0.79	83.1 ± 1.24	80.8 ± 1.43	78.3 ± 1.16	82.6 ± 1.58
Percent ≥ 120 mg %	5.1	2.8	5.4	4.2	2.4	6.8
Smoking						
Percent smokers	46.1	39.6	55.3	21.9	10.1	13.9
Never smoked	37.8	44.0	34.4	71.4	86.7	84.6
Ex-smokers	16.1	16.4	10.3	6.7	3.2	1.5
Exercise in leisure time						
Percent doing exercise	37.3	42.9	27.5	40.3	45.1	22.9

TABLE 101

Prevalence of Hyperglycaemia by Age & Sex

	30 - 39		40 - 49		50 - 60		All subjects	
	Males	Females	Males	Females	Males	Females	Males	Females
No. of Subjects	617	506	482	271	345	186	1444	963
No. with Hyperglycaemia	9	6	19	21	36	17	64	44
% with Hyperglycaemia	1.4	1.2	3.9	7.7	10.4	9.1	4.4	4.5

TABLE 102

Prevalence of angina and PMI and Fasting blood glucose level

Age and Sex		ANGINA						PMI					
		N	FBS < 120 mg% No.	%	N	FBS ≥ 120 mg% No.	%	N	FBS < 120 mg% No.	%	N	FBS ≥ 120 mg% No.	%
30 - 39	M	608	12	1.9	9	1	11.1	608	8	1.3	9	1	11.1
	F	500	4	0.8	6	0	0	500	2	0.4	6	0	0
40 - 49	M	463	16	3.4	19	2	10.5	463	9	1.9	19	1	5.3
	F	250	6	2.4	21	1	4.8	250	2	0.8	21	1	4.8
50 - 60	M	309	19	6.1	36	5	13.9	309	10	3.2	36	6	16.7
	F	169	7	4.1	17	1	5.9	169	1	0.6	17	1	5.9
Total	M	1380	47	3.4	64	8	12.5	1380	27	1.9	64	8	12.5
	F	919	17	1.8	44	2	4.5	919	5	0.5	44	2	4.5
Age-adjusted	M			3.4			11.6			1.9			10.5
	F			1.9			2.5			0.5			2.5

Significance of hyperglycaemia controlling for age and sex — P < 0.05 — P < 0.001

TABLE 103

Prevalence of ischaemic-type ECG changes and ischaemic-type ECG changes without symptoms and fasting blood glucose level

Age and Sex		Ischaemic-type ECG changes						Ischaemic-type ECG changes without symptoms					
		N	FBS < 120 mg% No.	%	N	FBS ≥ 120 mg% No.	%	N	FBS < 120 mg% No.	%	N	FBS ≥ 120 mg% No.	%
30 - 39	M	608	10	1.6	9	1	11.1	608	3	0.5	9	0	0
	F	500	5	1.0	6	0	0	500	2	0.4	6	0	0
40 - 49	M	463	21	4.5	19	3	15.7	463	12	2.6	19	2	10.5
	F	250	4	1.6	21	1	4.8	250	2	0.8	21	0	0
50 - 60	M	309	26	8.4	36	8	22.2	309	18	5.8	36	2	5.6
	F	169	9	5.3	17	3	17.6	169	6	3.5	17	1	5.9
Total	M	1380	57	4.1	64	12	18.7	1380	33	2.4	64	4	6.2
	F	919	18	1.9	44	4	9.1	919	10	1.1	44	1	2.3
Age-adjusted	M			4.2			15.3			2.5			4.8
	F			2.0			4.7			1.1			1.1
Significance of hyperglycaemia controlling for age and sex				$P < 0.01$						$P > 0.1$			

TABLE 104

Results of Water Analysis done in London and Amman

		Ca	P(as phosphate)	Mg	Fe	Zn	Pb	NO_2	NO_3
London Analysis	Deep well	75 ppm	< 0.1 ppm	33 ppm	<0.01 ppm	<0.01 ppm	< 0.1 ppm	<0.003 ppm	<0.5 ppm
	Spring water	72 ppm	< 0.1 ppm	21 ppm	<0.01 ppm	<0.01 ppm	< 0.1 ppm	<0.003 ppm	6 ppm
Amman Analysis	Deep well	67.3 ppm	< 0.1 ppm	33.1 ppm	<0.1 ppm	<0.1 ppm	<0.1 ppm	<0.01 ppm	<0.5 ppm
	Spring water	67.0 ppm	< 0.1 ppm	22.7 ppm	<0.1 ppm	<0.1 ppm	<0.1 ppm	<0.01 ppm	5 ppm

TABLE 105

PREVALENCE OF IHD & ORAL CONTRACEPTIVES

AGE		N	ANGINA		PMI		ECG		ECG without symptoms	
			No	%	No	%	No	%	No	%
30-39	Users	59	0	0	2	3.4	4	6.8	2	3.4
	Ex-users	90	1	1.1	0	0	0	0	0	0
	Never Used	357	3	0.8	0	0	1	0.3	0	0
40-49	Users	22	1	4.5	0	0	1	4.5	0	0
	Ex-users	70	0	0	2	2.9	2	2.9	1	1.4
	Never Used	170	6	3.3	1	0.6	2	1.1	1	0.6
50-60	Users	3	0	0	0	0	0	0	0	0
	Ex-users	69	5	7.2	1	1.4	7	10.1	5	7.7
	Never Used	114	3	2.6	1	0.8	5	4.4	2	1.7
Total	Users	84	1	1.2	2	2.4	5	6.0	2	2.4
	Ex-users	229	6	2.6	3	1.3	9	3.9	6	2.6
	Never Used	650	12	1.8	2	0.3	8	1.2	3	0.4
Age-adjusted	Users			1.3		1.8		4.8		1.8
	Ex-users			2.0		1.1		2.8		1.8
	Never Used			1.8		0.3		1.3		0.5

Significance of oral contraceptives controlling for age: $P>0.1$ | $0.05<P<0.1$ | $P<0.01$ | $P<0.05$

TABLE 106

Prevalence of Angina and Alcohol

Age and Sex		Drinkers			Ex-drinkers			Never drunk		
		N	Angina No.	%	N	Angina No.	%	N	Angina No.	%
30 - 39	M	25	2	8.0	31	2	6.5	561	9	1.6
	F	9	0	0	7	0	0	490	4	0.8
40 - 49	M	62	4	6.5	56	4	7.1	364	10	2.7
	F	17	2	11.8	7	0	0	247	5	2.0
50 - 60	M	54	6	11.1	55	4	7.3	236	14	5.9
	F	21	4	19.0	11	1	9.1	154	4	2.6
Total	M	141	12	8.5	142	10	7.0	1161	3	2.8
	F	47	6	12.8	25	1	4.0	891	12	1.3
Age-adjusted	M			8.2			6.9			3.0
	F			6.9			1.5			1.5

Significance of alcohol intake controlling for age and sex $P < 0.001$

TABLE 107

Prevalence of PMI and Alcohol

Age and sex		Drinkers			Ex-drinkers			Never drunk		
		N	PMI No.	%	N	PMI No.	%	N	PMI No.	%
30 - 39	M	25	2	8.0	31	1	3.2	561	6	1.1
	F	9	1	11.1	7	0	0	490	1	0.2
40 - 49	M	62	1	1.6	56	3	5.4	364	6	1.6
	F	17	1	5.9	7	0	0	247	2	0.8
50 - 60	M	54	2	3.7	55	5	9.1	236	9	3.8
	F	21	0	0	11	0	0	154	2	1.3
Total	M	141	5	3.5	142	9	6.3	1161	21	1.8
	F	47	2	4.3	25	0	0	891	5	0.5
Age-adjusted	M			4.8			5.3			1.9
	F			7.5			0			1.2

Significance of alcohol intake controlling for age and sex $P < 0.01$

TABLE 108

Prevalence of ischaemic-type ECG changes and alcohol

Age and Sex		Drinkers			Ex-drinkers			Never drunk		
		N	ECG No.	%	N	ECG No.	%	N	ECG No.	%
30 - 39	M	25	1	4.0	31	1	3.2	561	9	1.6
	F	9	1	11.1	7	0	0	490	4	0.8
40 - 49	M	62	4	6.5	56	4	7.1	364	16	4.4
	F	17	2	11.8	7	1	14.3	247	2	0.8
50 - 60	M	54	10	18.5	55	4	7.3	236	20	8.5
	F	21	4	19.0	11	0	0	254	8	5.2
Total	M	141	15	10.6	142	9	6.3	1161	45	3.8
	F	47	7	14.9	25	1	4.0	891	14	1.6
Age-adjusted	M			8.3			5.5			4.2
	F			12.8			4.0			1.6

Significance of alcohol intake controlling for age and sex $P < 0.001$

TABLE 109

Prevalence of ischaemic-type ECG changes without symptoms and alcohol

Age and Sex		Drinkers			Ex-drinkers			Never drunk		
		N	ECG without symptoms No.	%	N	ECG without symptoms No.	%	N	ECG without symptoms No.	%
30 - 39	M	25	0	0	31	1	3.2	561	9	1.6
	F	9	0	0	7	0	0	490	2	0.4
40 - 49	M	62	4	6.5	56	2	3.6	364	8	2.2
	F	17	0	0	7	1	14.3	247	1	0.4
50 - 60	M	54	7	13.0	55	0	0	236	13	5.5
	F	21	2	9.4	11	0	0	154	5	3.2
Total	M	141	11	7.8	142	2	1.4	1161	24	2.0
	F	47	2	4.3	25	1	4.0	891	8	0.9
Age-adjusted	M			5.3			1.2			2.3
	F			1.8			4.0			0.9

Significance of alcohol intake controlling for age and sex $P < 0.05$

TABLE 110

Multiple Logistic Regression of IHD Prevalence - Males

Variable	Angina (55 cases)		PMI (35 cases)		Ischaemic-type ECG (69 cases)		Ischaemic-type ECG without symptoms (37 cases)	
	Coefficient	t	Coefficient	t	Coefficient	t	Coefficient	t
Age (Years)	0.026	1.35	0.022	0.92	0.048	2.60**	0.079	3.15**
BMI (Weight/ Height^2)	-0.006	-0.50	-0.003	-0.26	0.004	0.72	0.006	1.13
Hypertension	0.859	2.59**	0.537	1.82	0.926	3.04**	0.979	2.53*
Serum Cholesterol (mg%)	0.0132	3.05**	0.017	3.21**	0.020	4.48***	0.014	2.75**
Hyperglycaemia	0.358	0.76	0.846	1.69	0.454	1.06	-0.541	-0.81
Cigarette Smoking	0.009	0.03	0.543	1.46	0.554	1.98*	0.411	1.14
Type A Behaviour	0.492	1.66	0.990	2.46*	0.287	1.04	0.318	0.89
Not doing exercise in leisure time	1.908	3.15**	1.795	2.42*	2.458	3.39***	1.708	2.32*
Type of bread+	-0.681	-1.52	-0.396	-0.73	-1.596	-3.38***	-1.432	-2.32*

* $P<0.05$
** $P<0.01$
*** $P<0.001$

+ White bread recoded 1.0
White & Brown " 1.5
Brown bread " 2.0

TABLE 111

Multiple Logistic Regression of IHD Prevalence - Females

Variable	Angina (19 cases)		PMI (7 cases)		Ischaemic-type ECG (22 cases)		Ischaemic-type ECG without symptoms (11 cases)	
	Coefficient	t	Coefficient	t	Coefficient	t	Coefficient	t
Age (Years)	0.042	1.27	0.001	0.05	0.031	0.87	0.087	1.81
BMI (Weight/ Height^2)	-0.069	-0.93	-0.005	-0.48	-0.006	-0.42	0.004	0.31
Hypertension Systolic pressure 160 or diastolic 95 mm Hg	1.783	2.87**	0.966	1.54	1.608	2.95**	0.339	0.43
Serum Cholesterol (mg%)	0.011	0.99	0.008	1.17	0.023	2.78**	0.0168	1.42
Hyperglycaemia FBS 120 mg%	-0.682	-0.60	0.190	0.24	0.140	0.18	-0.370	-0.32
Cigarette Smoking	1.387	2.67**	0.281	0.60	1.187	2.37*	1.264	1.90
Type A Behaviour	0.410	1.76	-0.464	-1.28	2.152	2.72**	1.184	1.42
Not doing exercise in leisure time	2.005	1.90	0.178	0.49	2.017	1.98*	1.150	1.06
Type of bread	0.560	0.75	-0.812	-2.93**	-1.665	-1.72	-1.435	-1.18

* $P < 0.05$
** $P < 0.01$

TABLE 112

AORTO-CORONARY BYPASS GRAFTS, 1979

	20 - 29	30 - 39	40 - 49	50 - 59	60 +
MALES	2	19	78	58	15
FEMALES	-	2	8	3	1

TABLE 113

Prevalence of ischaemic-type ECG abnormalities according to Minnesota Code criteria in Amman, Jamaica and selected U.S. and European surveys: males 40 - 49 years

ECG items	Minnesota Code	Jordan		Jamaica		U.S.A.				Europe							
		Amman (482)		Lawrence[a] Tavern (177)		Framingham[b] (779)		Tecumseh[c] (542)		Brussels[d] (631)		The Hague[d] (377)		Naples[d] (412)		Moscow[d] (169)	
		No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%	No.	%
Major Q/QS items	1_1 1_2	2 5	1.4	1 2	1.7	1 8	1.2	3 2	0.9	2 5	1.1	1 2	0.8	5 3	1.9	- 3	1.8
Minor Q/QS items	1_3	5	1.0	1	0.6	1	0.1	6	1.1	8	1.3	8	2.1	8	1.9	8	4.3
Left axis deviation	2_1	9	1.9	6	3.4	23	3.0	22	4.1								
High amplitude R waves	3_1	5	1.0	53	29.9	51	6.5	9	1.7	10	1.6	18	4.8	14	3.4	29	17.2
ST depression	4_1 4_2	3 3	1.6	1 5	4.5	5 7	2.2	2 4	1.7	1 6	1.3	- 3	1.3	1 9	2.7	- 1	0.6
	4_3	2		2		5		3		-		2		1		-	
T wave inversion	5_1 5_2	3 2	2.1	1 6	10.2	- 11	8.1	- 8	7.7	1 6	2.1	- -	1.6	- 7	4.6	- -	2.4
T wave flattening	5_3	5		11		52		34		6		6		12		4	
LBBB[e]	7_1	2	0.4	-	-	3	0.4	1.	0.2	-	-	-	-	1	0.2	-	-

a Miall et al (1972)
b Higgins et al (1965)
c Ostrander (1966)
d Rose et al (1968)
e Left bundle branch block

APPENDIX I

CONFIDENTIAL

Serial No. [| | |] 1-4

Punch Card No. [1] 5

Name:
(and maiden name) .....

Address: .....

Telephone Number: .....

Sex: Male [1] Female [2] 6

What was your date of birth: day [|] month [|] year [|] 7-12

Marital Status

Are you: single [1] married [2] divorced/separated [3] widowed [4] 13

Occupation

What is your occupation, Post Title ..... [ ] 14

	Yes	No	
Are you satisfied with your present occupation?	[1]	[2]	15
Are you overworked?	[1]	[2]	16
Do you work overtime?	[1]	[2]	17

Educational Status

What was the highest level of education you completed:

Code	The Subject	Wife/Husband	
	[ ]	[ ]	18-19
1 Never attended school			
2 Primary School			
3 Preparatory School			
4 Secondary School			
5 Vocational Training School			
6 Teachers Training Centre			
7 University			

Instruction: Enter appropriate code for level of education for subject and for spouse.

Smoking

1. (a) Do you smoke cigarettes now?

Yes regularly	1	20
No - IF 'NO' GO TO QUESTION 2(a)	2	
Occasionally (usually less than one cigarette per day)	3	

(b) Do you inhale?

Yes	1	21
No	2	

(c) What kinds of cigarettes do you smoke?

Manufactured with filters	Yes	1	22
	No	2	
Manufactured without filters	Yes	1	23
	No	2	
Hand-rolled	Yes	1	24
	No	2	

(d) How many manufactured cigarettes do you usually smoke per day? ___ 25-26

(e) About how many grams of tobacco do you use per week for rolling your own cigarettes? ___ 27-28

(f) What is the maximum number of cigarettes that you have smoked per day for as long as a year? ___ 29-30

(Record total number of manufactured and hand-rolled cigarettes, counting 1 g of tobacco as 1 cigarette.)

(g) How many cigarettes did you smoke per day a year ago? ___ 31-32

(h) How old were you when you began to smoke cigarettes? ___ 33-34

AFTER ASKING THIS QUESTION, GO TO QUESTION 3(a)

3. (a) Did you ever smoke cigarettes?

Yes, regularly	1	35
No, never - IF 'NO' GO TO QUESTION 3(a)	2	
Occasionally (usually less than one cigarette per day)	3	

(b) What is the maximum number of cigarettes you ever smoked per day for as long as a year? [ ] 36-37

(Record total number of manufactured and hand-rolled cigarettes, counting 1 g of tobacco as 1 cigarette.)

(c) Did you inhale?

Yes 1
No 2 [ ] 38

(d) How old were you when you began to smoke cigarettes? [ ] 39-40

(e) When did you stop smoking cigarettes? (Give year) [ ] 41-42

(f) Why did you stop?

Illness/doctor's orders [ ] 43
Health precaution [ ] 44
Cost [ ] 45
Other [ ] 46

3. (a) Have you ever smoked cigars?

No - IF 'NO' GO TO QUESTION 4(a) 1
Used to, but not now - IF 'NOT NOW' GO TO QUESTION 4(a) 2
Now smoke occasionally (less than one per day) 3
Now smoke regularly 4 [ ] 47

(b) About how many do you smoke per week? [ ] 48-49

(c) Do you inhale?

Yes 1
No 2 [ ] 50

4. (a) Have you ever smoked a pipe?

No 1
Used to, but not now 2
Now smoke a pipe occasionally (less than once a day) 3
Now smoke one regularly 4 [ ] 51

(b) About how many grams of tobacco to you smoke per week? [ ] 52-53

(c) Do you inhale?

Yes	1	54
No	2	

Alcohol Consumption

Do you currently drink alcohol?

Yes	1	55
No	2	

If 'NO', have you ever drunk alcohol?

Yes	1	56
No - IF 'NO' GO TO NEXT QUESTION	2	

If 'YES' what type?

Beer	Yes 1	Wine	Yes 1	Spirits	Yes 1	57-59
	No 2		No 2		No 2	

How many glasses do you usually drink per day?

Beer	1-3 1	Wine	1-3 1	Spirits	1-3 1	60-62
	4-8 2		4-8 2		4-8 2	
	9+ 3		9+ 3		9+ 3	

Diet and Dietary Habits

What kind of fat do you use?

	Animal	Vegetable	Both	
for cooking	1	2	3	63
for frying	1	2	3	64
for spreading	1	2	3	65

What kind of bread do you eat?

White	1	Brown	2	Both	3	66

How often do you eat

	Daily	3 times a week	Weekly	
Meat	1	2	3	67
Dry Beans	1	2	3	68
Green Beans	1	2	3	69
Citrus Fruit	1	2	3	70
Other Fruit (apples and bananas)	1	2	3	71

Card 3

Serial No. [ ] 1-4

Punch Card No. [2] 5

Psychosocial Factors

Instruction: Here is a list of several traits or qualities. For each one will you tell me whether each trait describes you very well, fairly well, somewhat, or not at all.

	Very Well	Fairly Well	Some-what	Not at all	
Being bossy or dominating	[1]	[2]	[3]	[4]	6
Having a strong need to excel (be best) in most things	[1]	[2]	[3]	[4]	7
Usually feeling pressed for time	[1]	[2]	[3]	[4]	8
Being hard driving and competitive	[1]	[2]	[3]	[4]	9
Eating too quickly	[1]	[2]	[3]	[4]	10

Instruction: Now we want to know how you have generally felt at the end of an average day in your regular line of work.

	Yes	No	
Have you often felt very pressed for time?	[1]	[2]	11
Has your work often stayed with you so that you were thinking about it after working hours?	[1]	[2]	12
Has your work often stretched you to the very limits of your energy and capacity?	[1]	[2]	13
Have you often felt uncertain, uncomfortable or dissatisfied with how well you were doing in your regular line of work?	[1]	[2]	14

Instruction: Finally,

Do you get quite upset when you have to wait for anything?	[1]	[2]	15

Have you suffered any of the following in the past 12 months?

	Yes	No	
Bereavement of close relative or friend,	[1]	[2]	16

1. Spouse
2. Brother/sister
3. Parent
4. 1st degree relative
5. Friend

Instruction: Enter appropriate code for relative or friend in coding box. [ ] 17

Have you suffered any of the following in the past 12 months?

	Yes	No	
Downgrading of employment	1	2	18
Serious financial loss	1	2	19
Serious family conflict	1	2	20
Other stressful events, specify	1	2	21

Now I would like to ask you some questions about your living conditions.

House

Of what material is your house or flat built?

Stone 1 Brick 2 Both brick and stone 3 Prefabricated 4 Other 5 specify 22

Do you own your house or flat or rent it?

Owned 1 Rented 2 23

How many rooms in your house (or, if you live in a flat, how many rooms in your flat) excluding bathroom and including kitchen? 24

How many people live in your house or flat? 25

Sources of Potable Water

What is your source of water for drinking and cooking?

Artesian Well	1	26
Spring	1	27
Collection Well	1	28
Bottled	1	29

Exercise in Leisure Time

Do you exercise in leisure time?

Yes 1 No 2 30

If 'YES' how often do you exercise?

Daily 1 Weekly 2 Occasionally 3 31

Do you exercise through:

	Yes	No	
Active (sport) recreation	1	2	32
Keep fit exercise	1	2	33
Heavy physical work	1	2	34
Gardening	1	2	35
Do-it-yourself	1	2	36
Vigorous getting about	1	2	37
Climbing 500+ stairs	1	2	38
Others:	1	2	39

PREVIOUS MEDICAL HISTORY

Section A: Chest Pain on Effort

1. Have you ever had any pain or discomfort in your chest? Yes 1 / No 2 — 40

   If 'NO' have you ever had any pressure or heaviness in your chest? Yes 1 / No 2 — 41

   If 'NO', proceed to Section C.

   If 'YES', ask next question. (If during the remainder of Section A an answer is recorded in a box marked *, proceed to Section B.)

2. Do you get it when you walk uphill or hurry? Yes 1 / No *2 / Never hurries or walks uphill 3 — 42

3. Do you get it when you walk at an ordinary pace on the level? Yes 1 / No 2 — 43

4. What do you do if you get it while you are walking? Stop of slow down 1 / Carry on *2 — 44

   (Record 'Stop or slow down' if subject carries on after taking nitroglycerine.)

5. If you stand still, what happens to it?

Relieved	1	45
Not relieved	2	

6. How soon?

10 minutes or less	1	46
More than 10 minutes	2	

7. Will you show me where it was? (Record all areas mentioned.)

Sternum (upper or middle)	1	47
Sternum (lower)	1	48
Left anterior chest	1	49
Left arm	1	50
Other	1	51

8. Do you feel it anywhere else? Yes 1, No 2 — 52

(If 'YES', record additional information above.)

9. Did you see a doctor because of this pain (or discomfort)? Yes 1, No 2 — 53

If 'YES', what did he say it was? ..... ICD Code ____ — 54-57

Section B: Possible Infarction

10. Have you ever had a severe pain across the front of your chest lasting for half an hour or more? Yes 1, No 2 — 58

If 'YES', ask question 11.

11. Did you see a doctor because of this pain? Yes 1, No 2 — 59

If 'YES', what did he say it was? ..... ICD Code ____ — 60-63

How many of these attacks have you had? ☐ 64

	Month	Year		Mins	
1st attack: date	☐☐	☐☐	Duration of pain	☐☐	65-70
2nd attack: date	☐☐	☐☐	Duration of pain	☐☐	71-76

Card 2

Serial No. ☐☐☐☐ 1-4

Punch Card No. 3 5

Section C: Intermittent Claudication

If an answer is recorded in a box marked *, no further questions need be asked.

12. Do you get pain in either leg on walking? Yes 1 / No *2 — 6

13. Does this pain ever begin when you are standing still or sitting? Yes *1 / No 2 — 7

14. In what part of your leg do you feel it?
Pain includes calf/calves 1 / Pain does not include calf/calves *2 — 8

If calves not mentioned, ask: Anywhere else?

15. Do you get it if you walk uphill or hurry? Yes 1 / No *2 / Never hurries or walks uphill 3 — 9

16. Do you get it if you walk at an ordinary pace on the level? Yes 1 / No 2 — 10

17. Does the pain ever disappear while you are walking? Yes *1 / No 2 — 11

18. What do you do if you get it when you are walking?
Stop or slow down 1 / Carry on *2 — 12

19. What happens if you stand still?

Relieved	[ ]	13
Not relieved	[2]	

20. How soon?

10 minutes or less	[1]	14
More than 10 minutes	[2]	

Have you ever been in hospital?

Yes	[1]	15
No	[2]	

If 'YES' please give the following information for each admission:

	Year	Diagnosis of nature of illness	ICD Code	
(1)	[ ][ ]	.....	[ ][ ][ ][ ]	16-21
(2)	[ ][ ]	.....	[ ][ ][ ][ ]	22-27
(3)	[ ][ ]	.....	[ ][ ][ ][ ]	2 -33
(4)	[ ][ ]	.....	[ ][ ][ ][ ]	34-39
(5)	[ ][ ]	.....	[ ][ ][ ][ ]	40-45

Has a doctor ever told you that your blood pressure was above normal?

Yes	[1]	46
No	[2]	

(If 'NO' go to next question)

When was the first time? (Give approximate year)

..... [ ][ ] 47-48

Have you ever had treatment for blood pressure?

Yes	[1]	49
No	[2]	

Are you taking treatment for it now?

Yes	[1]	50
No	[2]	

Have you ever had heart trouble, suspected or confirmed?

Yes	[1]	51
No	[2]	

(If 'NO' go to next question)

19. What happens if you stand still?

Relieved	1	13
Not relieved	2	

20. How soon?

10 minutes or less	1	14
More than 10 minutes	2	

Have you ever been in hospital?

Yes	1	15
No	2	

If 'YES' please give the following information for each admission:

	Year	Diagnosis of nature of illness	ICD Code	
(1)		.....		16-21
(2)		.....		22-27
(3)		.....		2 -33
(4)		.....		34-39
(5)		.....		40-45

Has a doctor ever told you that your blood pressure was above normal?

Yes	1	46
No	2	

(If 'NO' go to next question)

When was the first time? (Give approximate year)

..... 47-48

Have you ever had treatment for blood pressure?

Yes	1	49
No	2	

Are you taking treatment for it now?

Yes	1	50
No	2	

Have you ever had heart trouble, suspected or confirmed?

Yes	1	51
No	2	

(If 'NO' go to next question)

When was the first time? (give year)
..... [| |] 52-53

What was the diagnosis? ICD Code
..... [| | | |] 54-57

Did you attend hospital? Yes [1] 58
No [2]

Are you still attending a doctor for it? Yes [1] 59
No [2]

Has your wife, or any blood relative (living or dead), had heart trouble? Yes [1] 60
No [2]

(If 'NO' go to next question)

Please state:

Relationship	Age (roughly at diagnosis	Diagnosis ICD Code	
1. Spouse 2. Brother/sister 3. Parent 4. 1st degree relative			
..... [ ]	..... [	] ..... [	] 61-67
..... [ ]	..... [	] ..... [	] 68-74

Are you or have you been diabetic? Yes [1] 75
No [2]

Has your wife, or any blood relative (living or dead), had diabetes? Yes [1] 76
No [2]

If 'YES', please state relationship?

1. Spouse
2. Brother/sister
3. Parent
4. 1st degree relative

[ ] 77
[ ] 78

Card 4

Serial No. [| | |] 1-4

Punch Card No. [4] 5

For Women Only

Parity:

Have you ever been pregnant? Yes [1] No [2] 6

If 'YES', how many pregnancies have you had? 7

Oral Contraceptive Practice:

Have you ever used oral contraceptives? Yes [1] No [2] 8

If 'YES', are you currently using oral contraceptives? Yes [1] No [2] 9

If 'YES8, for how long? Less than 3 months [1] 3-12 months [2] More than 12 months [3] 10

If you do not use oral contraceptives now, did you ever use them? Yes [1] No [2] 11

If 'YES', for how long? Less than 3 months [1] 3-12 months [2] More than 12 months [3] 12

Menopausal State:

Are you still menstruating? Yes [1] No [2] 13

Physical Examination

Height ..... [| | .] cms 14-17

Weight ..... [| | .] kgms 18-21

(For office use) % Under/Over [| |] % 22-24

B.P. I S [| |] D [| |] Zero [|] 25-32

P.B. II S [| |] D [| |] Zero [|] 33-40

Plasma Cholesterol	☐☐☐ mg/100 ml	41-43
Fasting blood sugar	☐☐☐ mg/100 ml	44-46
ECG I		47
II		48
III		49
IV		50
V		51
VI		52
VII		53
VIII		54
IX		55
Summary		56
Interviewer Code	☐	57

APPENDIX 11

4-1 الرقم المتسلسل ☐☐☐☐

5 رقم البطاقة المثقبة [1]

الاسم (الاسم قبل الزواج) : .....

العنـــوان : .....

رقم الهـاتف : .....

6 الجنس : [1] ذكر / انثى [2]

12-7 تاريخ الميلاد : اليوم ☐☐ الشهر ☐☐ السنة ☐☐

13 الوضع العائلي : اعزب [1] متزوج [2] مطلق / منفصل [3] ارمل [4]

14 المهنة : لقب الوظيفة : .....☐

15 هل انت راض بعملك الحالي ؟ نعم [1] لا [2]

16 هل انت مرهق ؟ [1] [2]

17 هل تعمل وقتا اضافيا ؟ [1] [2]

الوضع التعليمي :

ما هو اعلى مستوى من التعليم وصلت اليه ؟

الشخص	الزوج / الزوجة
☐	☐

19-18 **الرمـز :**

(١) لم ادخل المدرسة ابدا
(٢) مدرسة ابتدائية
(٣) مدرسة اعدادية
(٤) مدرسة ثانوية
(٥) مدرسة التدريب المهني
(٦) مركز تدريب المعلمين
(٨) جـامعة

ملاحظة : ادخل الرمز المناسب لمستوى التعليم ، داخل المربع الشخص و زوجته / زوجة

التدخـــين :

١ - (أ) هل تدخن السجاير حاليا ؟

نعم ، وبصورة منتظمة — 1
لا (وفي هذه الحالة : انتقـل الى السؤال ٢ (أ)) — 2
احيانا (وعادة اقل من سيجارة واحدة يوميا) — 3 — 20

(ب) هل تستنشق دخان السجاير ؟

نعـــم — 1
لا — 2 — 21

(جـ) ماهي انواع السجاير التي تدخنها ؟

مصنوعة بفلتر — نعـم — 1 — 22
لا — 2

مصنوعة بدون فلتر — نعـم — 1 — 23
لا — 2

ملفوفـــة باليد — نعـم — 1 — 24
لا — 2

(د) كم عدد السجاير (المصنوعة) التي تدخنها عادة كل يوم ؟ — 26-25

(هـ) كم جراما من التبـغ تقريبا تستخدمها اسبوعيا في لف السجاير — 28-27

(و) ما هو اقصى عدد من السجاير قمت بتدخينه يوميا خلال عام ؟ — 30-29

« سجل مجموع عدد السجاير المصنوعة والملفوفة معا معتبرا ان كل جـرام من التبغ يساوي سيجـارة واحـدة »

ز) كم كان معدل السجاير التي قمت بتدخينها يوميا قبل عـام ؟ — 32-31

(ح) كم كان عمرك عندما بدأت بتدخين السجاير ؟ — 34-33

« بعد هذا السؤال ، انتقل الى السؤال ٣ دأ »

اذا كان الشخص يدخن حاليا انتقل بعد اجابة السؤال (١) الى السؤال ٣ (أ)

٢ - (أ) هل سبق ان دخنت السجاير ؟

نعم وبانتظام

لا ، ابدا « اذا كان «لا» انتقل الى السؤال ٣ « أ »

احيانا « عادة اقل من سيجارة يوميا »

(ب) كم كان اقصى عدد من السجاير التي دخنتها في اليوم خلال عام ؟

(سجل مجموع عدد السجاير المصنوعة والمفروطة معا، معتبرا ان الجرام الواحد من التبغ يساوي سيجارة واحدة)

« ج » هل كنت تستنشقها ؟

نعم

لا

« د » كم كان عمرك عندما بدأت في تدخين السجاير ؟

« هـ » متى اقلعت عن تدخين السجاير « اذكر السنة ؟

« ز » لماذا توقفت عن تدخين السجاير ؟

بسبب المرض او / اوامر الطبيب

كاحتياط صحي

المصروف

غير ذلك

٣ - هل سبق ان دخنت سيجارا ؟

- لا « انتقل الى السؤال ٤ « أ » »

- كنت ادخنه ولكن ليس حاليا « انتقل الى السؤال ٤ «أ» »

- حاليا ادخنه احيانا باقل من سيجار واحد في اليوم »

- حاليا ادخنه بانتظام

«ب» كم سيجارا تدخن تقريبا في الاسبوع ؟

« ج » هل تستنشق دخان السيجار ؟

نعم

لا

35 [1] [2] [3]

37-36 [ ][ ]

38 [1] [2]

40-39 [ ][ ]

42-41 [ ][ ]

43 [ ]

44 [ ]

45 [ ]

46 [ ]

47 [1] [2] [3] [4]

49-48 [ ][ ]

50 [1] [2]

٤ - « أ » هل سبق ان دخنت غليونا ؟

	[1]	لا
	[2]	كنت ادخنه ، ولكن ليس حاليا
51	[3]	ادخنه احيانا « اقل من مرة يوميا »
	[4]	ادخنه بانتظام حاليا

«ب» كم هي كمية التبغ التي تدخنها في الاسبوع تقريبا مقدرة بالجرامات ؟ [ ][ ] 53-52

«جـ» هل تستنشق دخان تبغ الغليون ؟

54	[1]	نعم
	[2]	لا

تعاطي المشروبات الروحية:

هل تتعاطى المشروبات الروحية حاليا ؟

55	[1]	نعم
	[2]	لا

اذا كان الجواب « لا » ، فهل سبق ان تعاطيتها ؟

56	[1]	نعم
	[2]	لا « ومن هنا ؟ انتقل الى السؤال التالي »

اذا كان الجواب « نعم » ، فما نوع الشراب ؟

59-57 بيرة : نعم [1] لا [2] نبيذ [1] [2] مشروبات روحية اخرى [1] [2]

كم كاسا تتناول في اليوم عادة ؟

	بيرة	نبيذ	مشروبات روحية
	[1] 3-1	[1] 3-1	[1] 3-1
62-60	[2] 8-4	[2] 8-4	[2] 8-4
	[3] ٩ فما فوق	[3] ٩ فما فوق	[3] ٩ فما فوق

الاكل وعادات الاكل :

ما هو نوع الدهن الذي تستعمله ؟

		حيواني	نباتي	كلاهما
63	الطبخ	1	2	3
64	الق...لي	1	2	3
65	للاكل	1	2	3

ما هو نوع الخبز الذي تتناوله ؟

66 ابيض [1] اسمر [2] كلاهما [3]

كم مرة تأكل المواد الغذائية التالية ؟

		يوميا	٣ مرات اسبوعيا	اسبوعيا
67	اللحم	1	2	3
68	الفاصوليا الناشفة	1	2	3
69	الفاصوليا الخضراء	1	2	3
70	الحمضيات	1	2	3
71	فواكه اخرى « تفاح وموز »	1	2	3

4-1 الرقم المتسلسل

5 رقم البطاقة المثقبة [2]

العوامل النفسية:

تعليمات : فيما يلي قائمة بصفات او ميزات عديدة ، ارجو ان تبين امام كل واحدة منها ما اذا كانت الصفة تنطبق عليك « ١ » كثيرا « جدا » « ٢ » بصورة متوسطة « جيدا » « قليلا » الى حد ما » « ٤ » لا تنطبق ابدا

		كثيرا	بدرجة متوسط	قليلا	ابدا
6	حب السيطرة او الترؤس	1	2	3	4

لديك الرغبة القوية للتفوق في معظم الامور	1	2	3	4	7
تشعر عادة بضغط العمل وضيق الوقت	1	2	3	4	8
طموح ومحب للمنافسة	1	2	3	4	9
تأكل الطعام بسرعة	1	2	3	4	10

تعليمات : والان ، نريد ان نعرف كيف كنت تشعر ، بصورة عامه في نهاية اليوم الذي تقوم فيه بعملك العادي

	نعم	لا	
هل كثيرا ما شعرت بانك مضغوط جداً بالنسبة للوقت ؟	1	2	11
هل كثيرا مايقي عملك معك بحيث كنت تفكر فيه بعد انتهاء ساعات العمل ؟	1	2	12
هل كثيرا ما تطلب عملك اقصى ما لديك من قدرة ونشاط ؟	1	2	13
هل كثيرا ما شعرت بانك غير متأكد وغير مرتاح او غير راض عن كم هو حسن اداؤك لعملك المنتظم ؟	1	2	14

تعليمات : واخيرا :

هل تشعر بانزعاج كبير عندما يكون عليك ان تنتظر شيئاً ما ؟	1	2	15
هل قاسيت اي مما يلي في الاثني عشر شهرا الماضية ؟			
فقدان قريب جدا او صديق	1	2	16

١ - الزوج / الزوجة
٢ - الاخ / الاخت
٣ - احد الوالدين
٤ - قريب جدا ، قرابة وثيقة ،
٥ - صديق

تعلـــيمات : ادخل الرمز المناسب للقريب او الصديق في المربع التالي

7 ☐

هل قاسيت من اي مما يلي في الاثني عشر شهرا الماضية ؟

تنزيل درجتك من الوظيفة	1	2	18
خسارة مالية جسيمة	1	2	19
خلاف عائلي خطير	1	2	20
احداث مجهدة « شدائد اخرى » اوضحها	1	2	21

البيت :

ما هي المادة المستخدمة في بناء بيتك او شقتك ؟

22 الحجر [1] الطوب [2] الطوب والحجر معا [3] مواد جاهزة [4]
غيرها [5] اوضح ذلك .....

هل الشقة او البيت الذي تسكنه ملك لك ام مستأجر ؟

23 ملك لي [1] مستأجر [2]

24 كم عدد الغرف في بيتـــك او شقتك بما في ذلـــك المطبخ وباستثناء الحمام ؟ ☐

25 كم عدد الاشخاص الذين يسكنون في بيتك او شقتك ☐

مصادر الماء الصالح للشرب :

ما هو مصدر الماء الصالح للشرب والطبخ

بئر ارتوازية	1	26
ينبوع ماء	1	27
مياه المطر المتجمعة في بئر	1	28
مياه معبأة في زجاجات	1	29

ممارسة الرياضة في اوقات الفراغ :

30 هل تمارس الرياضة في اوقات الفراغ نعـــم [1] لا [2]

اذا كان الجواب « نعم » فهل تمارسها .

31 يوميا [1] اسبوعيا [2] احيانا [3]

ما هو نوع الرياضة التي تمارسها .

		نعم	لا
32	انشطة رياضية	1	2
	تمارين رياضية للمحافظة على		
33	اللياقة البدنية	1	2
34	عمل بدني عنيف	1	2
35	العمل في الحديقة	1	2
36	اعمله بنفسك	1	2
		نعم	لا
37	المشي السريع الجهد	1	2
38	صعود ٥٠٠ درجة او اكثر	1	2
39	غير ذلك	1	2

التاريخ الطبي السابق

«القسم الاول»

الم صدري عند القيام بجهد

١ – هل سبق وان حدث لك اي مضايقة في الصدر

40 نعم [1] لا [2]

اذا لم يسبق ان شعرت بذلك فهل سبق ان شعرت بثقل اوانضغاط في الصدر

41 نعم [1] لا [2]

اذا كان الجواب « لا » ، انتقل الى القسم الثالث

اذا كان الجواب « نعم » انتقل الى السؤال التالي :

« اذا كان في احد المربعات التالية في هذا « القسم » علامة « * » انتقل الى القسم الثاني »

٢ – هل تشعر بالالم او المضايقة في الصدر عند المشي صاعداً مكاناً مرتفعاً او الاسراع في المشي .

42 نعم [1] لا [2]

لا يسرع في المشي او يمشي صاعداً مكانا مرتفعاً [3]

٣ – هل تشعر بالالم او الضيق في الصدر عنـــد المشي بخطى عادية على سطح مستو . ؟

43 نعم [1]
لا [2]

٤ – ماذا تفعل عندما يحدث الم او ضيق في الصدر وانت ماش ؟

44 اقف او اتباطأ [1]
استمر في المشي [2]

ملاحظة : سجل الجواب في المربع الاول « اقف او اتباطأ »
اذا كان المريض يستمر في المشي بعـد تناول حبوب النيتروجليسيرين

٥ – اذا وقفت ساكنا عند شعورك بالالم او الضيق في الصدر فماذا يحدث لك

45 يختفي [1]
لا يختفي [2]

٦ – متى يختفي الالم

46 بعد « ١٠ » دقائق او اقل [1]
بعد اكثر من « ١٠ » دقائق [2]

٧ – ارجو اطلاعي على مكان الالم « سجل جميع الامكنة »

47 القص الجزء العلوي او الاوسط [1]
48 القص الجزء الاسفل [1]
49 الناحية الامامية من الصدر [1]
50 الذراع الايسر [1]
51 اماكن اخرى [1]

52 ٨ – هل تشعر بالالم في اي مكان آخر . نعم [1] لا [2]
« اذا كان الجواب « نعم » سجل هـــذه الامكنة هنـا .....

٩ – هل كنت تزور الطبيب بسبب هذا الالم او الضيق ؟
53 نعم [1]
لا [2]

اذ كنت تزور الطبيب بسبب ذلك فماذا كان يخبرك عن سببه

57-54 ICD CODE [ ][ ][ ][ ]

القسم الثاني

احتمال وجود احتشاء في القلب

١٠ – هل سبق ان حدث لديك الم شديد في المنطقة الامامية من صدرك ، استمر مدة نصف ساعة او اكثر ؟

58 — نعم [1] — لا [2]

اذا كان الجواب « نعم » انتقل الى السؤال « ١١ »

١١ – هل زرت الطبيب بسبب ذلك الالم ؟

59 — نعم [1] — لا [2]

اذا كان الجواب « نعم » فماذا اخبرك عن سببه .

63 – 60 — ICD CODE [ ][ ][ ][ ]

64 — كم حدث لك من نوبات هذا الالم ؟ [ ]

70 – 65 — النوبة الاولى : الشهر [ ][ ] السنة [ ][ ] مدة الالم [ ][ ] دقائق

76 – 71 — النوبة الثانية : [ ][ ] مدة الالم [ ][ ]

4 – 1 — الرقم المتسلسل [ ][ ][ ][ ]

5 — رقم البطاقة المثقبة [3]

القسم الثالث

العرج المتقطع INTERMITTENT CLAUDICATION

ملاحظة: اذا وضع الجواب في المربع الموجودة فيه علامة (×) فلا موجب لاسئلة اخرى

١٢ – هل يحدث لديك الم في احدى الساقين عند المشي ؟

6 — نعم [1] — لا [2*]

١٣ – هل يحدث لديك هذا الالم عندما تكون واقفا او جالسا ؟

7 — نعم [1*] — لا [2]

١٤ – في اي جزء من ساقك تشعر بهذا الالم

8 — يشمل الالم بطة الساق / الساقين [1]

لا يشمل الالم بطة الساق / الساقين [2*]

« اذا لم يذكر بطة الساق / الساقين ، اسأل ، هل من مكان آخر تشعر فيه بالالم »

١٥ ـ هل يحدث لديك هذا الالم عند الصعود الى مكان مرتفع او الاسراع في المشي

9 [1] نعم [2]* لا [3] انني لا اصعد الى الاماكن المرتفعة او اسرع في المشي

١٦ ـ هل يحدث لديك هذا الالم اثناء المشي العادي على سطح مستو

10 [1] نعم [2] لا

١٧ ـ هل يختفي هذا الالم اثناء استمرارك في المشي ؟

11 [1]* نعم [2] لا

١٨ ـ ماذا تفعل عندما تشعر بهذا الالم اثناء المشي ؟

12 [1] اقف او اتباطأ [2]* استمر في المشي

١٩ ـ ماذا يحدث اذا توقفت عن المشي

13 [1] يختفي الالم [2] لا يختفي الالم

٢٠ ـ متى يختفي الالم

14 [1] خلال « ١٠ » دقائق او اقل [2] خلال اكثر من « ١٠ » دقائق

دخول المستشفى

١ ـ هل سبق ان ادخلت الى المستشفى

15 [1] نعم [2] لا

« اذا كان الجواب « نعم » اذكر لي المعلومات التالية لكل ادخال »

	العام	تشخيص المرض	ICD CODE
١ ـ	[ ][ ]	.....	21-16 [ ][ ][ ][ ]
٢ ـ	[ ][ ]	.....	27-22 [ ][ ][ ][ ]
٣ ـ	[ ][ ]	.....	33-29 [ ][ ][ ][ ]
٤ ـ	[ ][ ]	.....	39-34 [ ][ ][ ][ ]
٥ ـ	[ ][ ]	.....	45-40 [ ][ ][ ][ ]

ارتفاع ضغط الدم الشرياني

هل اخبرك الطبيب بأن ضغط دمك اعلى من المستوى الطبيعي ؟

46 نعم [1]
لا [2]

« اذا كان الجواب « لا » ، انتقـــل الى السؤال التالي
« عن القلب »

متى اخبرك ذلك لاول مرة ؟

48-47 اذكر السنـة بالتقريب ..... [ ][ ]

هل سبق ان عولجت لارتفاع الضغط

49 نعم [1]
لا [2]

هل تتناول حاليا علاجا لارتفاع الضغط

50 نعم [1]
لا [2]

مـــرض القلـب

هل سبق ان اصبت بمرض في القلب سواء اكان ذلك مشتبها به ام متأكد منه .

51 نعم [1]
لا [2]

« اذا كان الجواب « لا » انتقل الى السؤال التالي »

53-52 متى حدثت هذه الاصابة للمرة الاولى . اذكر السنة [ ][ ]

ICD CODE

57-54 ماذا كان تشخيص هذه الاصابة [ ][ ][ ][ ]

هل ادخلت الى المستشفى ؟

58 نعم [1]
لا [2]

هل ما زلت تزور الطبيب بسببها

59 نعم [1]
لا [2]

هل لدى زوجتك حاليا او لدى اي قريب لك قرابة دم وثيقة او كان لديه ، ان كان متوفيا . مرض بالقلب ؟

60 نعم [1]
لا [2]

اذا كان الجواب لا انتقل الى السؤال التالي

ارجو ذكر المعلومات التالية عن الاقارب المرضى :

نوع القرابة	العمر تقريبا عند التشخيص	التشخيص
١ – الزوج / الزوجة	.....	.....
٢ – الاخ / الاخت	.....	.....
٣ – الاب / الام	.....	.....
٤ – قريب قرابة دم وثيقة	.....	.....

67–61 ICD CODE ☐☐☐☐ ☐☐ ☐

74–68 ☐☐☐☐ ☐☐ ☐

داء السكري

75 هل انت مصاب او كنت مصابا بداء السكري ؟ نعم [1] لا [2]

76 هل لدى زوجتك او لدى اي قريب لك قرابة دم ، او كان لدية اذا كان متوفيا ، داء السكري ؟ نعم [1] لا [2]

اذا كان الجواب نعم اذكر نوع القرابة

١ – زوج / زوجة

٢ – اخ / اخت

77 ٣ – اب / ام ☐

78 ٤ – قريب قرابة دم وثيقة ☐

4-1 الرقم المتسلسل ☐☐☐☐

5 رقم البطاقة المثقبة [4]

للنساء فقط

الحمل :

6 هل سبق ان حملت
نعم [1]
لا [2]

7 اذا كان الجواب نعم فكم مرة حملت ☐☐

حبوب منع الحمل

8 هل سبق ان تناولت حبوب منع الحمل
نعم [1]
لا [2]

اذا كان الجواب نعم فهل انت حاليا تأخذين هذه الحبوب ؟

9
نعم [1]
لا [2]

اذا كان الجواب نعم فمنذ متى تأخذينها

10 اقل من ٣ شهور [1] ٣-١٢ شهرا [2] اكثر من ١٢ شهرا [3]

اذا انت لا تأخذين حبوب منع الحمل ، فهل سبق ان كنت تأخذينها ؟

11
نعم [1]
لا [2]

اذا كان الجواب نعم فكم كانت فترة استعمالها ؟

12 اقل من ٣ اشهر [1] ٣-١٢ شهرا [2] اكثر من ١٢ شهرا [3]

سن اليأس

13 هل ما زال الطمث يحدث لديك
نعم [1]
لا [2]

4-1 الرقم المتسلسل ☐☐☐☐

5 رقم البطاقة المثقبة [4]

للنساء فقط

الحمل :

6 هل سبق ان حملت
[1] نعم
[2] لا

7 اذا كان الجواب نعم فكم مرة حملت ☐☐

حبوب منع الحمل

8 هل سبق ان تناولت حبوب منع الحمل
[1] نعم
[2] لا

9 اذا كان الجواب نعم فهل انت حاليا تأخذين هذه الحبوب ؟
[1] نعم
[2] لا

10 اذا كان الجواب نعم فمنذ متى تأخذينها
[1] اقل من ٣ شهور [2] ٣ - ١٢ شهرا [3] اكثر من ١٢ شهرا

11 اذا انت لا تأخذين حبوب منع الحمل ، فهل سبق ان كنت تأخذينها ؟
[1] نعم
[2] لا

12 اذا كان الجواب نعم فكم كانت فترة استعمالها ؟
[1] اقل من ٣ اشهر [2] ٣ - ١٢ شهرا [3] اكثر من ١٢ شهرا

سن اليأس

13 هل ما زال الطمث يحدث لديك
[1] نعم
[2] لا

الفحص الجسمي

17-14 الطول .....

21-18 الوزن .....

24-22 اقل / اكثر الاستعمال المكتب ٪

32-25 ضغط الدم ١ – انقباضي ارتخائي قراءة صفر

40-33 ٣ – انقباضي ارتخائي قراءة صفر

43-41 الكولسترول في البلازما ملغم / ١٠٠ مل

46-44 سكر الدم على الريق ملغم / ١٠٠ مل

47 تخطيط القلب الكهربائي ١ – .....

48 ٢ – .....

49 ٣ – .....

50 ٤ – .....

51 ٥ – .....

52 ٦ – .....

53 ٧ – .....

54 ٨ – .....

55 ٩ – .....

56 موجز التخطيط .....

57 رمز الذي اجرى المقابلة

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